DISSERTATION

on

A STUDY OF THE PLACENTAL CHANGES IN HIGH RISK PREGNANCY

submitted in partial fulfillment of

requirements for

MD DEGREE EXAMINATION

BRANCH-III PATHOLOGY

THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI

TIRUNELVELI MEDICAL COLLEGE

TIRUNELVELI

MAY- 2019

CERTIFICATE

This is to certify that the dissertation titled **"A STUDY OF THE PLACENTAL CHANGES IN HIGH RISK PREGNANCY",** is a bonafide work done by **Dr.M.R.ALMAS BANU**, Post Graduate Student, Department of Pathology, Tirunelveli Medical College, Tirunelveli – 627011, in partial fulfilment of the university rules and regulations for the award of MD DEGREE in PATHOLOGY BRANCH-III, under my guidance and supervision, during the academic period from 2016 to 2019.

Place: Tirunelveli

Date:

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CERTIFICATE

I hereby certify that this dissertation entitled "**A STUDY OF THE PLACENTAL CHANGES IN HIGH RISK PREGNANCY**" is a record of work done by **Dr.M.R.ALMAS BANU**, in the Department of Pathology, Tirunelveli Medical College, Tirunelveli, during his postgraduate degree course period from 2016- 2019. This work has not formed the basis for previous award of any degree.

Place: Tirunelveli

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DECLARATION

I solemnly declare that the dissertation titled **"A STUDY OF THE PLACENTAL CHANGES IN HIGH RISK PREGNANCY"** was done by me at Tirunelveli Medical College,Tirunelveli – 627011, during the period 2016 to 2019 under the guidance and supervision of **Prof.K.SHANTARAMAN, MD**, to be submitted to The Tamil Nadu Dr. M.G.R. Medical University towards the partial fulfilment of requirements for the award of MD DEGREE in PATHOLOGY BRANCH-III.

Place : Tirunelveli

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PROTOCOL TITLE: A STUDY OF THE PLACENTAL CHANGES IN HIGH RISK PREGNANCY
PRINCIPAL INVESTIGATOR: Dr.ALMAS BANU.M.R. MBBS.,
DESIGNATION OF PRINCIPAL INVESTIGATOR: PG STUDENT
DEPARTMENT & INSTITUTION: TIRUNELVELI MEDICAL COLLEGE, TIRUNELVELI

Dear, Dr.ALMAS BANU.M.R., MBBS., The Tirunelveli Medical College Institutional Ethics Committee (TIREC) reviewed and discussed your application during the IEC meeting held on 10.03.2017

THE FOLLOWING DOCUMENTS WERE REVIEWED AND APPROVED

1. TIREC Application Form ☐
2. Study Protocol ☐
3. Department Research Committee Approval ☐
4. Patient Information Document and Consent Form in English and Vernacular Language ☐
5. Investigator's Brochure ☐
6. Proposed Methods for Patient Accrual Proposed ☐
7. Curriculum Vitae of the Principal Investigator ☐
8. Insurance /Compensation Policy ☐
9. Investigator's Agreement with Sponsor ☐
10. Investigator's Undertaking ☐
11. DCGI/DGFT approval ☐
12. Clinical Trial Agreement (CTA) ☐
13. Memorandum of Understanding (MOU)/Material Transfer Agreement (MTA) ☐
14. Clinical Trials Registry-India (CTRI) Registration ☐

THE PROTOCOL IS APPROVED IN ITS PRESENTED FORM ON THE FOLLOWING CONDITIONS

1. The approval is valid for a period of 2 year/s or duration of project whichever is later
2. The date of commencement of study should be informed
3. A written request should be submitted 3weeks before for renewal / extension of the validity
4. An annual status report should be submitted.
5. The TIREC will monitor the study
6. At the time of PI's retirement/leaving the institute, the study responsibility should be transferred to a person cleared by HOD
7. The PI should report to TIREC within 7 days of the occurrence of the SAE. If the SAE is Death, the Bioethics Cell should receive the SAE reporting form within 24 hours of the occurrence.
8. In the events of any protocol amendments, TIREC must be informed and the amendments should be highlighted in clear terms as follows:
   a) The exact alteration/amendment should be specified and indicated where the amendment occurred in the original project. (Page no. Clause no. etc.)
   b) The PI must comment how proposed amendment will affect the ongoing trial. Alteration in the budgetary status, staff requirement should be clearly indicated and the revised budget form should be submitted.
   c) If the amendments require a change in the consent form, the copy of revised Consent Form should be submitted to Ethics Committee for approval. If the amendment demands a re-look at the toxicity or side effects to patients, the same should be documented.
   d) If there are any amendments in the trial design, these must be incorporated in the protocol, and other study documents. These revised documents should be submitted for approval of the IEC, only then can they be implemented.
   e) Approval for amendment changes must be obtained prior to implementation of changes.
   f) The amendment is unlikely to be approved by the IEC unless all the above information is provided.
   g) Any deviation/violation/waiver in the protocol must be informed.

STANDS APPROVED UNDER SEAL

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CERTIFICATE – II

This is certify that this dissertation work title **"A STUDY OF THE PLACENTAL CHANGES IN HIGH RISK PREGNANCY"** of the candidate **Dr.M.R.ALMAS BANU** with registration Number **201613301** for the award of **M.D.** Degree in the branch of **PATHOLOGY (III).** I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion page and result shows **8 percentage** of plagiarism in the dissertation.

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ABBREVIATION

PC – post conception

hPL – human placental lactogen

BP – blood pressure

IGT - Impaired Glucose Tolerance

GDM - Gestational Diabetes Mellitus

CS – caesarean section

Cm - Centimetre

IUGR - Intrauterine growth restriction

SGA – Small for gestation

FGR – fetal growth restriction

CD – cluster of differentiation

RH - retroplacental hematoma

AFI - amniotic fluid index

PG –prostaglandin

ACE – Angiotensin converting enzyme

VUE - villitis of unknown etiology

IUD – intra uterine death

DiDi - Dichorionic diamnionic

DiMo - Monochorionic diamnionic

MoMo - Monochorionic monoamnionic

SLE - Systemic lupus erythematosus

ANA - antinuclear antibody

DPX – Dibutyl phthalate xylene

Pih – pregnancy induced hypertension

Hb – haemoglobin

INDEX

S.NO	PATHOLOGY NUMBER	IP.NO	AGE	GESTATIONAL CODE	RISK FACTOR	GESTATIONAL AGE	PLACENTAL WEIGHT	CORD	INFARCT > 5%	TENNEY PARKAR CHANGE	DISTAL VILLOUS HYPOPLASIA	RETROPLACENTAL HAEMORRHAGE	MATURITY OF VILLI FOR GESTATION	FIBRINOID NECROSIS	INTRAVILLOUS FIBRIN
1	514/2017	5100	37	G2P1L1	PLACENTA INCRETA	38 WEEKS	450 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
2	546/2017	13278	28	G2P1P1	COMPLETE MOLE	14 WEEKS	160 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	IMMATURE	ABSENT	ABSENT
3	553/2017	13656	28	G3P2L1	PARTIAL MOLE	13 WEEKS	100 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	IMMATURE	ABSENT	ABSENT
4	622/2017	14667	26	G2P1L1	PIH 140/100 mm Hg	38 WEEKS	450 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
5	632/2017	17606	22	G2P1L1	PARTIAL MOLE	15 WEEKS	130 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	IMMATURE	ABSENT	ABSENT
6	648/2017	15080	25	G3P2L2	PLACENTA PREVIA	38 WEEKS	450 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
7	996/2017	223781	27	PRIMI	PARTIAL MOLE	18 WEEKS	200 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	IMMATURE	ABSENT	ABSENT
8	1012/2017	26055	35	G3P2L2	PLACENTA PREVIA	38 WEEKS	480 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
9	1015/2017	25547	27	G3P2L2	PARTIAL MOLE	14 WEEKS	110 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	IMMATURE	ABSENT	ABSENT
10	1031/2017	28768	26	G2P1L1	PIH BP : 160/100 mm Hg	37 WEEKS	500 GRAMS	CENTRAL	PRESENT	ABSENT	ABSENT	ABSENT	MATURE	PRESENT	PRESENT
11	1272/2017	32807	20	PRIMI	PARTIAL MOLE	15 WEEKS	140 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	IMMATURE	ABSENT	ABSENT
12	1344/2017	35767	21	PRIMI	PIH BP : 150/90 mm Hg	37 WEEKS	500 GRAMS	CENTRAL	PRESENT	PRESENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
13	1546/2017	38165	48	G4P3L2A1	PARTIAL MOLE	15 WEEKS	130 GRAMS	CENTRAL	PRESENT	ABSENT	ABSENT	ABSENT	IMMATURE	ABSENT	ABSENT
14	1645/2017	40742	24	G2P1L1	PARTIAL MOLE	13 WEEKS	110 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	IMMATURE	ABSENT	ABSENT
15	1727/2017	43692	35	G3P2L2	INTRAUTERINE DEATH	22 WEEKS	150 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
16	1734/2017	41809	30	G3P2L2	PLACENTA ACCRETA	38 WEEKS	420 GRAMS	CENTRAL	PRESENT	ABSENT	ABSENT	ABSENT	IMMATURE	PRESENT	PRESENT
17	1831/2017	38978	25	G2P1L1	PLACENTA ACCRETA, PRI	39 WEEKS	480 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
18	1856/2017	43895	25	G2P1L1	PLACENTA PREVIA	37 WEEKS	450 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
19	1926/2017	47413	28	G2P2L1	ANAEMIA	39 WEEKS	500 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
20	2138/2017	54887	25	PRIMI	PARTIAL MOLE	19 WEEKS	200 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	IMMATURE	ABSENT	ABSENT
21	2156/2017	56788	27	G2P1L1	OLIGOHYDRAMNIOS	38 WEEKS	550 GRAMS	ECCENTRIC	ABSENT	ABSENT	ABSENT	ABSENT	HYPERMATURE	PRESENT	PRESENT
22	2275/2017	58088	35	G3P2L1A1	INTRAUTERINE DEATH	23 WEEKS	220 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	IMMATURE	ABSENT	ABSENT
23	2351/17	59828	27	G2P1L1	PIH, IUD	25 WEEKS	200 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	PRESENT
24	2367/2017	60978	29	PRIMI	GESTATIONAL DIABETES	39 WEEKS	450 GRAMS	ECCENTRIC	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
25	2371/2017	59608	30	G3P1L1A1	PARTIAL MOLE	18 WEEKS	190 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	IMMATURE	ABSENT	ABSENT
26	2379/2017	61084	27	G2P1L1	PLACENTA PREVIA	36 WEEKS	450 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
27	2433/2017	62158	25	PRIMI	INTRAUTERINE DEATH	22 WEEKS	180 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	PRESENT	PRESENT
28	2494/2017	64009	25	PRIMI	PRE ECLAMPSIA	36 WEEKS	550 GRAMS	CENTRAL	PRESENT	ABSENT	ABSENT	ABSENT	MATURE	PRESENT	PRESENT
29	2495/2017	63285	21	PRIMI	PRE ECLAMPSIA	37 WEEKS	525 GRAMS	CENTRAL	PRESENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
30	2612 /2017	67596	20	PRIMI	PARTIAL MOLE	20 WEEKS	250 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	IMMATURE	ABSENT	ABSENT
31	2470 /2017	64913	23	PRIMI	ABRUPTION	30 WEEKS	300 GRAMS	N0 COIL	PRESENT	ABSENT	ABSENT	PRESENT	HYPERMATURE	PRESENT	PRESENT
32	2536/2017	58789	22	PRIMI	ANAEMIA	38 WEEKS	500 GRAMS	CENTRAL	PRESENT	ABSENT	ABSENT	ABSENT	MATURE	PRESENT	PRESENT
33	2574/2017	66921	35	G4P3L2A1	PLACENTA ACCRETA	38 WEEKS	480 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT
34	2647/2017	70506	25	PRIMI	GESTATIONAL DIABETES	38 WEEKS	480 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	PRESENT	PRESENT
35	2648/2017	68357	24	PRIMI	PIH, SLE	36 WEEKS	400 GRAMS	MARGINAL	PRESENT	PRESENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
36	2765/2017	74700	27	G2P1L1	OLIGOHYDRAMNIOS	38 WEEKS	550 GRAMS	ECCENTRIC	ABSENT	ABSENT	ABSENT	ABSENT	HYPERMATURE	PRESENT	PRESENT
37	2674/2017	70674	30	G3P2L2	ANAEMIA	38 WEEKS	490 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
38	2713/2017	71967	31	G3P2L2	PARTIAL MOLE	19 WEEKS	200 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	IMMATURE	ABSENT	ABSENT
39	2764/2017	73569	31	G2P1L1	ABRUPTION	35 WEEKS	520 GRAMS	CENTRAL	PRESENT	ABSENT	ABSENT	PRESENT	MATURE	PRESENT	PRESENT
40	2755/2017	74890	30	G2P1L1	OLIGOHYDRAMNIOS	38 WEEKS	500 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	PRESENT	ABSENT
41	2785/2017	76845	24	G3P2L2	ANAEMIA	40 WEEKS	480 GRAMS	CENTRAL	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT
42	2799/2017	70659	30	G3P2L2	PLACENTA ACCRETA, PRI	38 WEEKS	480 GRAMS	ECCENTRIC	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
43	2812/2017	78314	33	G2P1L1	OLIGOHYDRAMNIOS	39 WEEKS	450 GRAMS	CENTRAL	PRESENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT

S.NO	PATHOLOGY NUMBER	IP.NO	AGE	GESTATIONAL CODE	RISK FACTOR	GESTATIONAL AGE	PLACENTAL WEIGHT	CORD	INFARCT > 5%	TENNEY PARKAR CHANGE	DISTAL VILLOUS HYPOPLASIA	RETROPLACENTAL HAEMORRHAGE	MATURITY OF VILLI FOR GESTATION	FIBRINOID NECROSIS	INTRAVILLOUS FIBRIN
44	2818/2017	78408	25	PRIMI	HYPOTHYROID, IUD	25 WEEKS	180 GRAMS	CENTRAL	PRESENT	ABSENT	ABSENT	ABSENT	HYPERMATURE	PRESENT	PRESENT
45	2819/2017	78524	25	PRIMI	ECLAMPSIA	36 WEEKS	500 GRAMS	CENTRAL	PRESENT	ABSENT	PRESENT	ABSENT	MATURE	PRESENT	PRESENT
46	2900/2017	79032	23	PRIMI	PARTIAL MOLE	18 WEEKS	220 GRAMS	ECCENTRIC	PRESENT	ABSENT	ABSENT	ABSENT	IMMATURE	PRESENT	ABSENT
47	2901/2017	80032	24	PRIMI	COMPLETE MOLE	16 WEEKS	140 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	IMMATURE	ABSENT	ABSENT
48	2956/2017	81551	21	PRIMI	PARTIAL MOLE	19 WEEKS	200 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	IMMATURE	ABSENT	ABSENT
49	3011/2017	8499	33	G3P2L2	PLACENTA ACCRETA, PRI	37 WEEKS	500 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
50	3014/2017	84200	30	G3P1L1A1	TAKAYASU ARTERITIS	13 WEEKS	150 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
51	3089/2017	87345	21	PRIMI	ANAEMIA	37 WEEKS	480 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	PRESENT	PRESENT
52	3091/2017	84577	26	PRIMI	OLIGOHYDRAMNIOS	39 WEEKS	350 GRAMS	CENTRAL	MULTIPLE	ABSENT	ABSENT	ABSENT	MATURE	PRESENT	PRESENT
53	3185/2017	86398	25	PRIMI	ANAEMIA	38 WEEKS	490 GRAMS	ECCENTRIC	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
54	3204/2017	90192	34	G3P2L2A1	INTRAUTERINE DEATH	29 WEEKS	300 GRAMS	CENTRAL	PRESENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
55	3281/17	90946	23	PRIMI	ANAEMIA	39 WEEKS	490 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
56	3307/2017	91846	23	PRIMI	ANAEMIA	38 WEEKS	480 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	PRESENT	PRESENT
57	16/2018	90045	23	G2P1L1	ANAEMIA	40 WEEKS	525 GRAMS	CENTRAL	PRESENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
58	17/2018	95579	29	G2P1L1	GESTATIONAL DIABETES	39 WEEKS	750 GRAMS	CENTRAL	PRESENT	ABSENT	ABSENT	ABSENT	MATURE	PRESENT	PRESENT
59	38/2018	1342	23	PRIMI	OLIGOHYDRAMNIOS	38 WEEKS	450 GRAMS	CENTRAL	PRESENT	ABSENT	ABSENT	ABSENT	MATURE	PRESENT	PRESENT
60	85/2018	2010	30	G3P2L2	PLACENTA PERCRETA,PR	37 WEEKS	450 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
61	88/2018	94293	30	G3P2L2	PLACENTA INCRETA, PRE	37 WEEKS	450 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
62	103/2108	1316	29	G3P2L1A1	ANAEMIA	38 WEEKS	480 GRAMS	ECCENTRIC	PRESENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
63	117/2018	5595	21	PRIMI	ECLAMPSIA	39 WEEKS	500 GRAMS	CENTRAL	PRESENT	ABSENT	PRESENT	ABSENT	HYPERMATURE	PRESENT	PRESENT
64	118/2018	5352	36	G3P2L1D1	INTRAUTERINE DEATH	21 WEEKS	350 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	IMMATURE	ABSENT	ABSENT
65	137/2018	9984	23	G3P1L1A1	RH NEGATIVE, IUD	21 WEEKS	250 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
66	138/2018	7084	27	PRIMI	INTRAUTERINE DEATH	30 WEEKS	300 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	PRESENT	PRESENT
67	281/2018	7557	21	PRIMI	INTRAUTERINE DEATH	37 WEEKS	360 GRAMS	N0 COIL	PRESENT	ABSENT	ABSENT	ABSENT	MATURE	PRESENT	PRESENT
68	282/2018	11718	22	G2P1L1	PARTIAL MOLE	26 WEEKS	550 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	IMMATURE	ABSENT	ABSENT
69	291/2018	9584	24	PRIMI	PARTIAL MOLE	18 WEEKS	200 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	IMMATURE	ABSENT	ABSENT
70	396/2018	14463	23	PRIMI	PIH, PARTIAL MOLE	29 WEEKS	600 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	HYPERMATURE	ABSENT	ABSENT
71	398/2018	14525	28	PRIMI	PIH BP 140/100 mm Hg	38 WEEKS	500 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
72	410/2018	11967	31	G3P2L2	PLACENTA INCRETA, PRE	37 WEEKS	480 GRAMS	CENTRAL	PRESENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
73	421/2018	13827	29	G2P1L1	INTRAUTERINE DEATH	21 WEEKS	220 GRAMS	ECCENTRIC	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
74	596/2018	20721	27	G2P1L1	GESTATIONAL DIABETES	38 WEEKS	500 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT
75	601/2018	19852	19	PRIMI	PARTIAL MOLE	20 WEEKS	220 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	IMMATURE	ABSENT	ABSENT
76	655/2018	22065	30	G3P2L1A1	PLACENTA ACCRETA	40 WEEKS	450 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
77	700/2018	24126	26	G2P1L1	OLIGOHYDRAMNIOS	36 WEEKS	460 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
78	707/2018	24839	29	G2P1L1	ANAEMIA	39 WEEKS	480 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
79	708/2018	24774	29	PRIMI	ANAEMIA	38 WEEKS	525 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
80	721/2018	27366	30	G2P2L2	ANAEMIA	39 WEEKS	500 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
81	723/2018	27965	23	PRIMI	PIH BP 150/100 mm Hg	38 WEEKS	490 GRAMS	ECCENTRIC	PRESENT	ABSENT	ABSENT	ABSENT	MATURE	PRESENT	PRESENT
82	810/2018	29024	26	PRIMI	IUGR	37 WEEKS	400 GRAMS	CENTRAL	ABSENT	ABSENT	PRESENT	ABSENT	DYSMATURE	ABSENT	ABSENT
83	820/2018	30456	29	G5P2L1A3	ANAEMIA	35 WEEKS	420 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
84	851 /2018	32964	27	G4P3L3	PLACENTA INCRETA, PRE	38 WEEKS	500 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
85	941/2018	33209	30	G3P2L2	PLACENTA ACCRETA	37 WEEKS	480 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
86	954/2018	33779	26	PRIMI	OLIGOHYDRAMNIOS	36 WEEKS	450 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
87	964/2018	34378	25	G3P3L1A1	PLACENTA ACCRETA, PRI	37 WEEKS	460 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT

S.NO	PATHOLOGY NUMBER	IP.NO	AGE	GESTATIONAL CODE	RISK FACTOR	GESTATIONAL AGE	PLACENTAL WEIGHT	CORD	INFARCT > 5%	TENNEY PARKAR CHANGE	DISTAL VILLOUS HYPOPLASIA	RETROPLACENTAL HAEMORRHAGE	MATURITY OF VILLI FOR GESTATION	FIBRINOID NECROSIS	INTRAVILLOUS FIBRIN
88	976 /2018	22486	23	PRIMI	OLIGOHYDRAMNIOS	39 WEEKS	480 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
89	977 /2018	31767	24	PRIMI	GESTATIONAL DIABETES,	37 WEEKS	775 GRAMS	TWO	PRESENT	ABSENT	ABSENT	ABSENT	DYSMATURE	PRESENT	PRESENT
90	978 /2018	25072	29	PRIMI	OLIGOHYDRAMNIOS	39 WEEKS	575 GRAMS	ECCENTRIC	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
91	979/2018	24303	23	PRIMI	ANAEMIA	40 WEEKS	500 GRAMS	CENTRAL	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT
92	1079/2018	39158	21	PRIMI	COMPLETE MOLE	14 WEEKS	140 GRAMS	CENTRAL	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	ABSENT
93	1340/2018	46659	21	G2P1L1	COMPLETE MOLE	13 WEEKS	150 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	IMMATURE	ABSENT	ABSENT
94	1382/2018	46587	22	PRIMI	PIH, MULTIPLE DAMC	37 WEEKS	750 GRAMS	TWO	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	ABSENT
95	1388/2018	49550	33	G3P2L1A1	IUGR	39 WEEKS	400 GRAMS	CENTRAL	PRESENT	ABSENT	ABSENT	ABSENT	MATURE	PRESENT	PRESENT
96	1436/2018	48396	24	PRIMI	OLIGOHYDRAMNIOS	37 WEEKS	450 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	PRESENT	PRESENT
97	1495/2018	48894	18	PRIMI	INTRAUTERINE DEATH	30 WEEKS	300 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	MATURE	ABSENT	PRESENT
98	1551/2018	49053	24	PRIMI	INTRAUTERINE DEATH	23 WEEKS	120 GRAMS	CENTRAL	ABSENT	ABSENT	ABSENT	ABSENT	DYMATURE	PRESENT	PRESENT

INCREASED PERIVILLOUS FIBRIN DEPOSITION	INTERVILLOUS HAEMORRHAGE	SYNCYTIAL KNOTS	SMOOTH MUSCLE PROLIFERATION OF FEEDING VESSELS	CALCIFICATION	ATHEROSCLEROSIS	THROMBOSIS	VILLOUS ENLARGEMENT	VILLOUS EDEMA	TROPHOBLASTIC ATYPIA	VILLOUS SHAPE	CISTERN FORMATION	VILLOUS VASCULARITY		TROPHOBLAST INVASION INTO MYOMETRIUM
ABSENT	ABSENT	NORMAL	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NORMAL	ABSENT	PRESENT	ABSENT	PRESENT
ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	ROUND	ABSENT	ABSENT	CIRCUMFERENTIAL	ABSENT
ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ENLARGED, HYDROPIC	PRESENT	ABSENT	SCALLOPED	MINIMAL	REDUCED MARKEDLY	PRESENT, FOCAL	ABSENT
PRESENT	PRESENT	NORMAL	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ENLARGED	PRESENT	ABSENT	SCALLOPED	PRESENT	ABSENT	FOCAL	ABSENT
ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	SCALLOPED	PRESENT	ABSENT	FOCAL	ABSENT
ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	SCALLOPED	PRESENT	REDUCED	PRESENT	ABSENT
ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	SCALLOPED	PRESENT	REDUCED MARKEDLY	PRESENT	ABSENT
PRESENT	PRESENT	NORMAL	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	SCALLOPED	PRESENT	ABSENT	FOCAL	ABSENT
PRESENT	PRESENT	NORMAL	PRESENT	PRESENT	PRESENT	PRESENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	SCALLOPED	PRESENT	REDUCED MARKEDLY	PRESENT, FOCAL	ABSENT
ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	SCALLOPED	PRESENT	REDUCED	PRESENT	ABSENT
ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
PRESENT	ABSENT	NORMAL	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	PRESENT
ABSENT	PRESENT	NORMAL	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	PRESENT
ABSENT	ABSENT	NORMAL	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
ABSENT	ABSENT	NORMAL	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	SCALLOPED	PRESENT	PRESENT	PRESENT	ABSENT
PRESENT	ABSENT	INCREASED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	NORMAL	ABSENT	ABSENT
PRESENT	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	NORMAL	ABSENT	ABSENT
ABSENT	PRESENT	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	SCALLOPED	PRESENT	ABSENT	PRESENT	ABSENT
ABSENT	ABSENT	NORMAL	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
PRESENT	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
PRESENT	PRESENT	NORMAL	PRESENT	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
PRESENT	PRESENT	NORMAL	PRESENT	PRESENT	PRESENT	PRESENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	SCALLOPED	ABSENT	REDUCED	PRESENT	ABSENT
PRESENT	PRESENT	PRESENT	ABSENT	ABSENT	ABSENT	PRESENT	FOCAL	FOCAL	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
PRESENT	PRESENT	INCREASED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
ABSENT	ABSENT	NORMAL	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	PRESENT
PRESENT	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	INCREASED	ABSENT	ABSENT
ABSENT	PRESENT	INCREASED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
PRESENT	ABSENT	INCREASED	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
PRESENT	PRESENT	NORMAL	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	PRESENT	PRESENT, FC	SCALLOPED	PRESENT	PRESENT	PRESENT	ABSENT
PRESENT	PRESENT	NORMAL	ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
ABSENT	ABSENT	NORMAL	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
ABSENT	ABSENT	NORMAL	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
ABSENT	ABSENT	NORMAL	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	PRESENT
ABSENT	ABSENT	NORMAL	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT

INCREASED PERIVILLOUS FIBRIN DEPOSITION	INTERVILLOUS HAEMORRHAGE	SYNCYTIAL KNOTS	SMOOTH MUSCLE PROLIFERATION OF FEEDING VESSELS	CALCIFICATION	ATHEROSCLEROSIS	THROMBOSIS	VILLOUS ENLARGEMENT	VILLOUS EDEMA	TROPHOBLASTIC ATYPIA	VILLOUS SHAPE	CISTERN FORMATION	VILLOUS VASCULARITY		TROPHOBLAST INVASION INTO MYOMETRIUM
PRESENT	PRESENT	INCREASED	PRESENT	PRESENT	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
PRESENT	PRESENT	INCREASED	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	SCALLOPED	PRESENT	ABSENT	PRESENT	ABSENT
ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	PRESENT	PRESENT, DI	IRREGULAR	PRESENT	ABSENT	CIRCUMFERENTIAL	ABSENT
ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	PRESENT	PRESENT, FO	SCALLOPED	PRESENT	PRESENT	PRESENT	ABSENT
ABSENT	ABSENT	NORMAL	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ADHERENT
ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ENLARGED	PRESENT	ABSENT	ROUND	ABSENT	ABSENT	ABSENT	ABSENT
PRESENT	ABSENT	INCREASED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	NORMAL	ABSENT	ABSENT
PRESENT	PRESENT	NORMAL	PRESENT	PRESENT	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
PRESENT	PRESENT	NORMAL	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	ABSENT	ABSENT	ABSENT
PRESENT	PRESENT	INCREASED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
PRESENT	ABSENT	NORMAL	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	INCREASED	ABSENT	ABSENT
PRESENT	PRESENT	NORMAL	PRESENT	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	HYPERVASCULAR	ABSENT	ABSENT
PRESENT	PRESENT	NORMAL	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	NORMAL	ABSENT	ABSENT
PRESENT	PRESENT	NORMAL	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	PRESENT, FU
ABSENT	ABSENT	NORMAL	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	SCALLOPED	ABSENT	PRESENT	ABSENT	PRESENT
PRESENT	PRESENT	NORMAL	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	INCREASED	ABSENT	ABSENT
PRESENT	PRESENT	INCREASED	PRESENT	ABSENT	PRESENT	PRESENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	INCREASED	ABSENT	ABSENT
ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
PRESENT	PRESENT	ILL DEFINED	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	POOR	ABSENT	ABSENT
ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	HYPOVASCULAR	ABSENT	ABSENT
PRESENT	PRESENT	INCREASED	PRESENT	PRESENT	PRESENT	PRESENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	REDUCED	ABSENT	ABSENT
ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ENLARGED	PRESENT	ABSENT	IRREGULAR	PRESENT	PRESENT	FOCAL	ABSENT
ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	SCALLOPED	PRESENT	ABSENT	PRESENT	ABSENT
ABSENT	ABSENT	INCREASED	ABSENT	ABSENT	PRESENT	PRESENT	PRESENT	PRESENT	ABSENT	SCALLOPED	PRESENT	PRESENT	FOCAL	ABSENT
ABSENT	ABSENT	NORMAL	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT
PRESENT	ABSENT	NORMAL	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	PRESENT
PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
ABSENT	ABSENT	NORMAL	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	PRESENT	ABSENT	SCALLOPED	PRESENT	PRESENT	PRESENT	ABSENT
ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ADHERENT
ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
ABSENT	PRESENT	NORMAL	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
PRESENT	PRESENT	NORMAL	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	HYPOVASCULAR	ABSENT	ABSENT
PRESENT	PRESENT	INCREASED	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	INCREASED	ABSENT	ABSENT
ABSENT	ABSENT	PRESENT	MINIMAL	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	PRESENT
ABSENT	ABSENT	NORMAL	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
ABSENT	PRESENT	NORMAL	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT
ABSENT	ABSENT	NORMAL	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT

INCREASED PERIVILLOUS FIBRIN DEPOSITION	INTERVILLOUS HAEMORRHAGE	SYNCYTIAL KNOTS	SMOOTH MUSCLE PROLIFERATION OF FEEDING VESSELS	CALCIFICATION	ATHEROSCLEROSIS	THROMBOSIS	VILLOUS ENLARGEMENT	VILLOUS EDEMA	TROPHOBLASTIC ATYPIA	VILLOUS SHAPE	CISTERN FORMATION	VILLOUS VASCULARITY		TROPHOBLAST INVASION INTO MYOMETRIUM
PRESENT	PRESENT	NORMAL	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
PRESENT	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	FOCAL	FOCAL	ABSENT	ROUND	ABSENT	HYPERVASCULAR	ABSENT	ABSENT
ABSENT	PRESENT	NORMAL	PRESENT	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	NORMAL	ABSENT	ABSENT
PRESENT	PRESENT	NORMAL	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	NORMAL	ABSENT	ABSENT
ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	PRESENT	PRESENT, DI	SCALLOPED	PRESENT	ABSENT	CIRCUMFERENTIAL	ABSENT
PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	PRESENT	PRESENT	PRESENT, DI	ROUND	PRESENT	MINIMAL	CIRCUMFERENTIAL	ABSENT
PRESENT	PRESENT	NORMAL	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
PRESENT	PRESENT	INCREASED	ABSENT	ABSENT	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
PRESENT	PRESENT	INCREASED	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	NORMAL	ABSENT	ABSENT
PRESENT	PRESENT	ABSENT	PRESENT	ABSENT	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
PRESENT	PRESENT	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ROUND	ABSENT	PRESENT	ABSENT	ABSENT
										SCALLOPED				

INTRODUCTION

Placenta, the fetal diary is a unique, autonomous and transient organ. It ensures maternal-fetal exchange of gases and nutrients. Placenta represents "the black box" of maternal-fetal-placenta system evolution. Apart from its nutritional function, it is an endocrine organ producing chorionic gonadotropin, oestrogens and progesterone (Ryan, 1962). It also has the capacity to degrade and conjugate steroids (Villee, 1962).[1] Common yet life threatening complications of Pregnancy like Gestational Diabetes, Hypertension, Anaemia and Intra uterine growth retardation result in both macroscopic as well as microscopic changes in the placenta. Hence study of the placenta will give a valuable insight in cases of adverse foetal outcome.

HIGH RISK PREGNANCY

A high risk pregnancy is one in which some condition put the mother, the developing fetus, or both at higher-than-normal risk for complications during or after the pregnancy and birth. Major causes of high risk pregnancies are hypertensive disorders, anaemia, intrauterine growth retardation, intrauterine death, oligohydramnios, hydatidiform mole, gestational diabetes mellitus, placenta accrete syndrome.

AIMS AND OBJECTIVES

• To observe & study the various gross morphological changes in the placentas of high risk mothers.

• To observe & study the various histopathological changes that occurs in placentas of high risk mothers

REVIEW OF LITERATURE

Development

Prelacunar Stage: Day 1 to 8 Post Conception[2](PC)

The period from conception to day 8 post conception is called prelacunar stage. After fertilization, the zygote develops into a blastocyst, a flattened vesicle composed of cells ranging from107 and 256 cells. The cells of the outer wall are the trophoblast, which surround the blastocystic cavity (Figure 1). At the eighth day of development, the blastocyst is partially embedded in the endometrial stroma. The trophoblast which is facing the maternal tissue are fused together to form syncytiotrophoblast. The remaining inner layer of blastocystic cavity is composed of mononucleated cells called cytotrophoblast. Syncytiotrophoblast loses its generative capacity during fusion. Cytotrophoblast acts as stem cell for the trophoblasts, with subsequent fusion to form synticytiotrophoblast.

Lacunar stage

On day 8 PC, small vacuoles appear in the syncytiotrophoblastic mass. The vacuoles grow and become confluent, forming a system of lacunae which are separated from each other by bands of syncytiotrophoblast, called trabeculae (figure 2).

The syncytiotrophoblastic mass and the lacunar system expand circumferentially over the entire blastocystic surface. By day 12 PC, the blastocyst is deeply implanted in the uterine epithelium. The cytotrophoblastic cells extend into the trabeculae and, by day 13 PC, reach the trophoblastic shell, eventually coming into contact with the endometrium. Trophoblastic proliferation and syncytial fusion start at the implantation pole, making the trophoblast thicker here. This area of preferential growth is later transformed into the placental disk. The opposing thinner trophoblastic circumference only initially attempts to establish the same structure. Eventually, it atrophies and becomes the smooth chorion, or the chorion laeve.

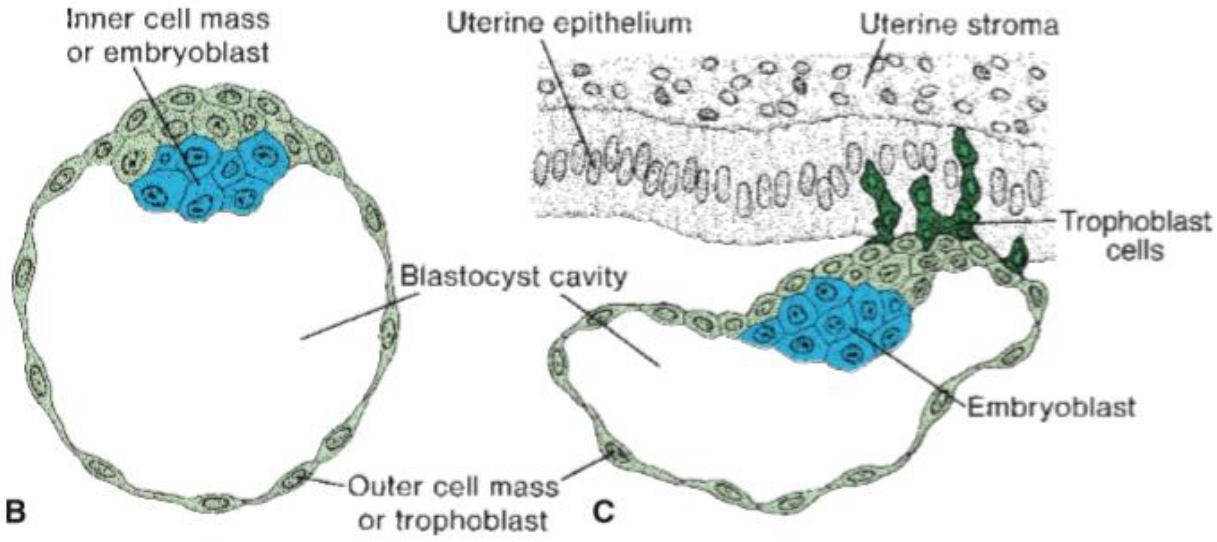


Figure 1: Pre lacunar stage

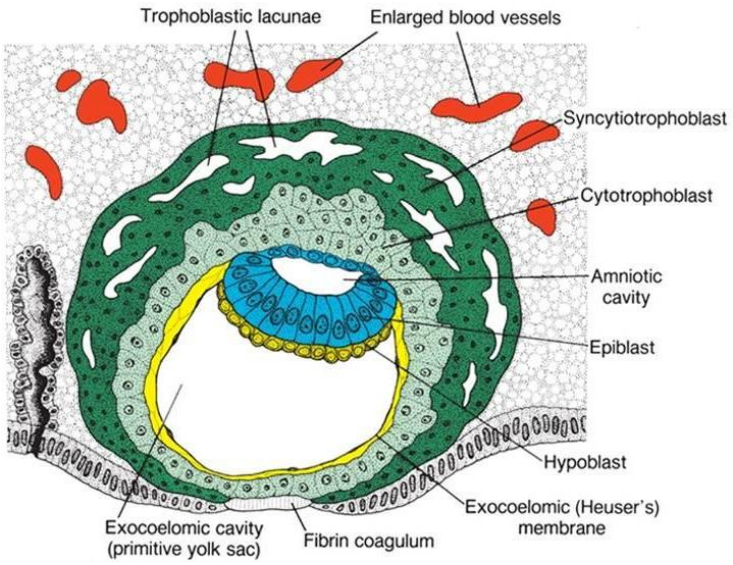


Figure 2: Lacunar stage

Early Villous Stage: Day 13 to 28 Post Conception

By the beginning of third week, Primary villi are formed which is composed only of an outer layer of syncytiotrophoblast and a core of cytotrophoblast. Subsequently, secondary villi consisting of an outer layer of syncytiotrophoblast, an inner layer of cytotrophoblast, and a core of connective tissue develops. By the end of the third week, mesodermal cells in the core of the villus begin to differentiate into blood cells and small blood vessels, forming the villous capillary system which is known as a tertiary villus.[3] Clusters of cytotrophoblast surrounded by an incomplete layer of syncytiotrophoblast persist as cell columns. They are places of longitudinal growth of the anchoring villi as well as sources of extravillous trophoblast. Focally, the villous tips of free-floating villi may not be invaded by villous mesenchyme, and these become the trophoblastic cell islands. The fetal and

maternal bloodstreams are always separated by the placental barrier which is composed of syncytiotrophoblast, cytotrophoblast, basal lamina, connective tissue, and fetal endothelium. [4]

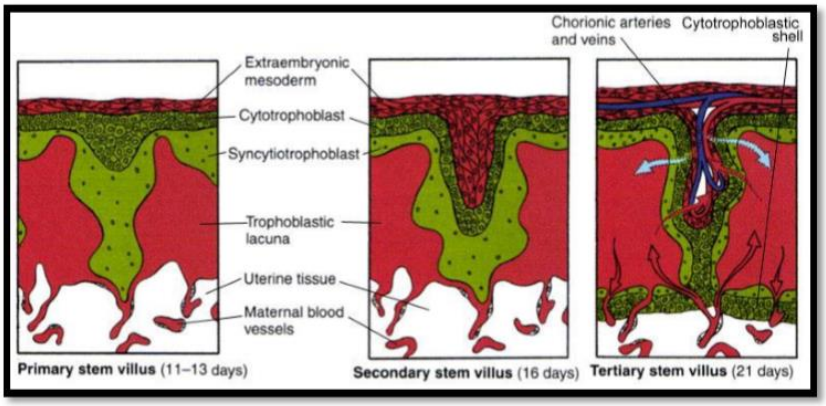


Figure 3 Formation of chorionic villi

The decidua at the implantation site is the decidua basalis. The decidual layer over the abembryonic pole is the decidua capsularis.[2] The remaining decidua which is without contact with the blastocyst (i.e., on the opposite uterine wall), is the parietal decidua or decidua vera. There is no true fusion between the decidua capsularis and the decidua vera.[4]

The Second Month and Beyond[4]

The connective tissue layer of the chorionic plate becomes more densely fibrotic and fibrous tissue extends into the villous stems. Subsequently, the tertiary villi undergo a complex process of differentiation

that results in various villous types which differ from each other in structure and function. With maturation, the syncytiotrophoblast is reduced in thickness and the cytotrophoblast becomes rarified. The mean villous diameter decreases, and the fetal capillaries are more numerous and closer to the villous surfaces.

GENERAL GROSS AND MICROSCOPIC FEATURES OF PLACENTA

The normal term placenta measures 15–20 cm in diameter and 1.5–3 cm in thickness, and weighs 450–600 g. The main components are the umbilical cord, membranes (amnion and chorion), villous parenchyma, and maternal decidual tissue [5.]

Umbilical Cord

The mean cord length at term is about 50 to 60 cm.[6] A short cord is considered a length less than 32 cm; this has been linked to conditions with restricted fetal movement. A long cord is defined as >100 cm and a short cord as <30 cm. There may be as many as 40 spiral twists in the cord, as well as false knots and true knots. Short umbilical cords may be associated with adverse perinatal outcomes such as fetal growth restriction, congenital malformations, intrapartum distress, and a twofold risk of death (Krakowiak and associates, 2004).[7] Short cords should be diagnosed by the pathologist

with caution and only in cases where the entire length of the cord has truly been measured. Long cords seem to be more susceptible to knots, cord entanglement, torsion, acute prolapse, and occlusion by compression.[8] Umbilical cord insertion can be central, eccentric, marginal (battledore), and velamentous (membranous) insertion.(figure 4) Central and eccentric account for more than 90% of term placenta.[9]

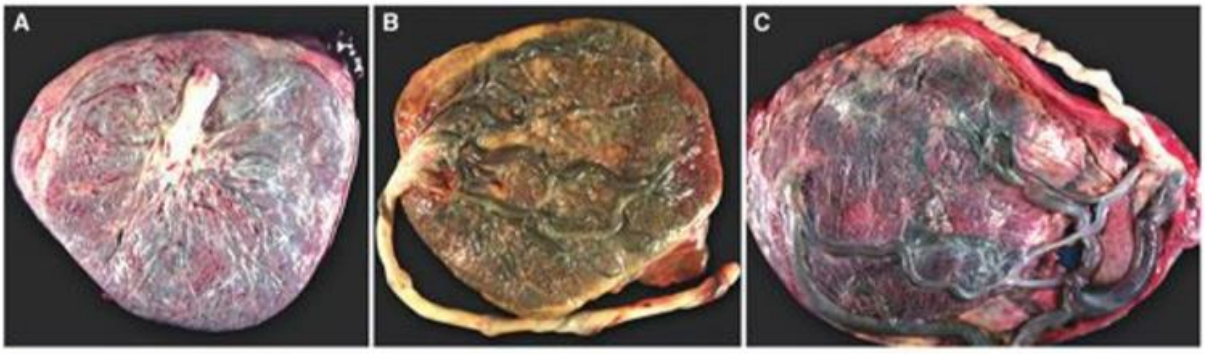


Figure : 4 Attachment of umbilical cord

A - normal, B – marginal, C – velamentous

It has an outer layer of amniotic epithelium, which becomes stratified at the fetal end. The bulk of the cord is made up of highly mucoid connective tissue known as *Wharton's jelly*. Embedded within its substance are the umbilical vessels, represented by two arteries and a single vein. The arteries have a double-layered muscular wall but no internal elastic lamina. The vein has a larger diameter and a thinner wall, consisting of a single layer of circular smooth muscle, and an internal elastic lamina.

Membranes

The *placental membranes* consist of the amnion and chorion. The *amnion*, which represents the innermost covering of the amniotic cavity, is lined by a single layer of flat epithelial cells resting on a basement membrane. *Squamous metaplasia* is common in them, especially near the insertion of the cord. The *chorion* is composed of a connective tissue membrane that carries the fetal vasculature. Its inner aspect is bounded by the outer layer of the amnion, and the outer aspect is associated with villi that sprout from the surface. The chorion associated with the membrane is called *chorion laeve* and is distinguished from the *chorion frondosum* located in the placental disk.

Placental disk

The placental disk normally has a round to oval shape. Each villus has an outer syncytiotrophoblast layer of basophilic cells that have a brush border of microvilli at their surface to increase the absorptive area. Immediately below the syncytiotrophoblast layer are the cytotrophoblast cells figure 5 (arrowheads), which are abundant in the early stages of pregnancy. Each villus has a basal lamina separating the epithelium from the subjacent core of loose connective tissue. Within the connective tissue core of the villi are branches of the umbilical arteries and vein. Fibroblasts and phagocytic cells (Hofbauer's cells) are also found there.[10]

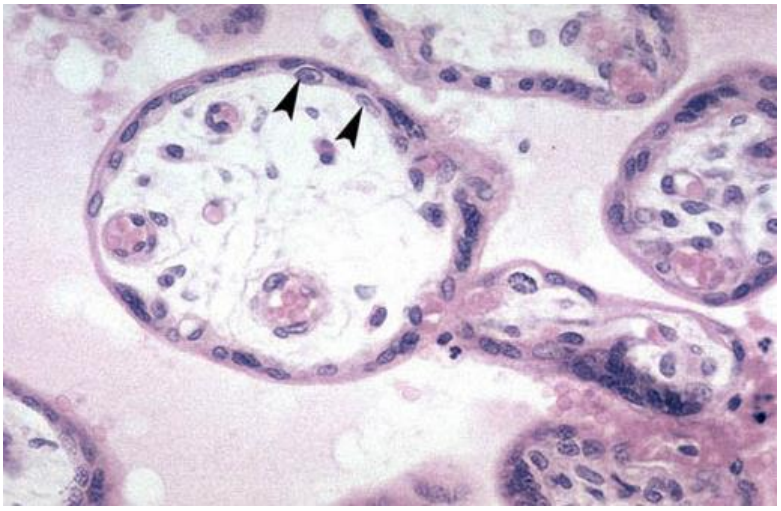


Figure 5 : Cytotrophoblast

In the term placenta, the cytotrophoblast is inconspicuous, and the syncytiotrophoblast is clumped in the form of ‘syncytial knots’.

The third trophoblastic type is represented by the *intermediate trophoblast,* also known as interstitial extravillous trophoblast and X cells.[11]This type is present in the villi and in the membranes but is particularly numerous in the extravillous region that forms the deepest structural component of the implantation site. The most distinctive immunohistochemical property of these cells is a strong reactivity for hPL

Structure of villi

The ramification of villous trees can be subdivided into segments which differ in their calibre, stromal structure, vessel structure, and position

within the villous tree. Five villous types have been described (figure 6). They are

- Stem villi
- Mature intermediate villi
- Terminal villi
- Immature intermediate villi
- Mesenchymal villi

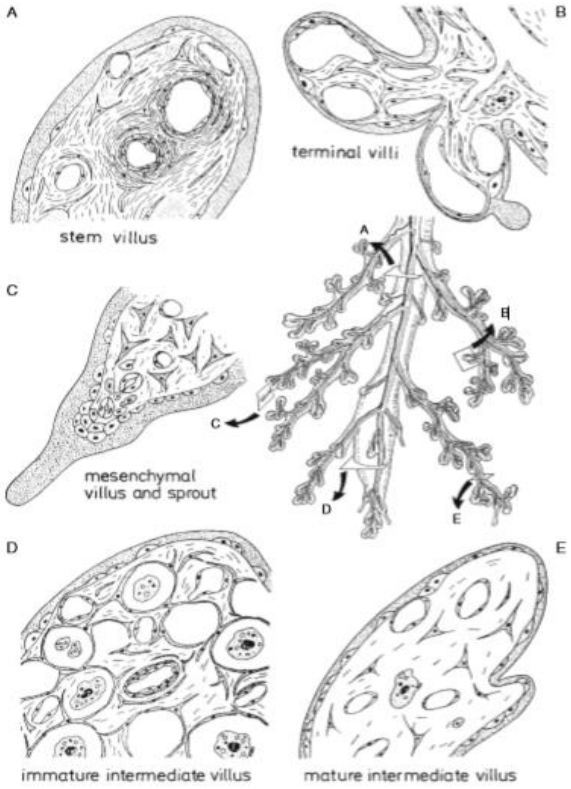


Figure 6: Structure of villi

The mesenchymal villi are the first generation of the tertiary villi and are the precursors from which all other villous types arise. Immature intermediate villi develop from maturation of mesenchymal villi during the first two trimesters and are later transformed into stem villi (Figure 5 A). The mature intermediate villi derive from mesenchymal villi during the third trimester and are later transformed into terminal villi (Figure 5 B). Thus, both types of “intermediate” villi are transitions from mesenchymal to mature villi. These intermediate villi topographically lie between the centrally located stem villi and the most peripheral terminal villi.

Table 1 [4]

TYPES OF VILLI

Villous type	% volume at term	Size	Characteristic features
Mesenchymal villi	<1	120–250micron	Primitive stroma, thick trophoblastic cover, few vessels
Immature intermediate villi	5 to 10	100–200 micron	Reticular stroma with fluid-filled stromal channels
Stem villi	20 to 25	150–300 micron	Dense fibrotic stroma and myofibroblastic perivascular sheath, large vessels
Mature intermediate villi	25	80–150micron	Dense, cellular stroma with >50% villi capillaries
Terminal villi	40 to 50	60micron	>50% capillaries

Microscopic structure of placenta
• Chorionic villi of various types
• Intervillous space
• Nonvillous parts of the placenta chorionic plate, cell islands, cell columns, placental septa, basal plate, marginal zone, and fibrinoid deposits

Nonvillous parts of the placenta

These structures do not participate in maternofetal exchange, but have mechanical and metabolic functions. Irrespective of their location and structure, the nonvillous parts of the placenta have the same three basic components,

• Extravillous trophoblast,

• Fibrinoid,

• Decidua

Extravillous Trophoblast.

Also called "X-cells", placental site trophoblast, and intermediate trophoblast. These are round to polygonal cells that are present singly or in groups. They tend to have pleomorphic, hyperchromatic, or irregular nuclei and intermediate trophoblast cytology is similar to that of surrounding decidualized stromal cells but the trophoblast have amphophilic cytoplasm, nuclei are slightly larger, irregular, and hyperchromatic. (Figure 7).[12]

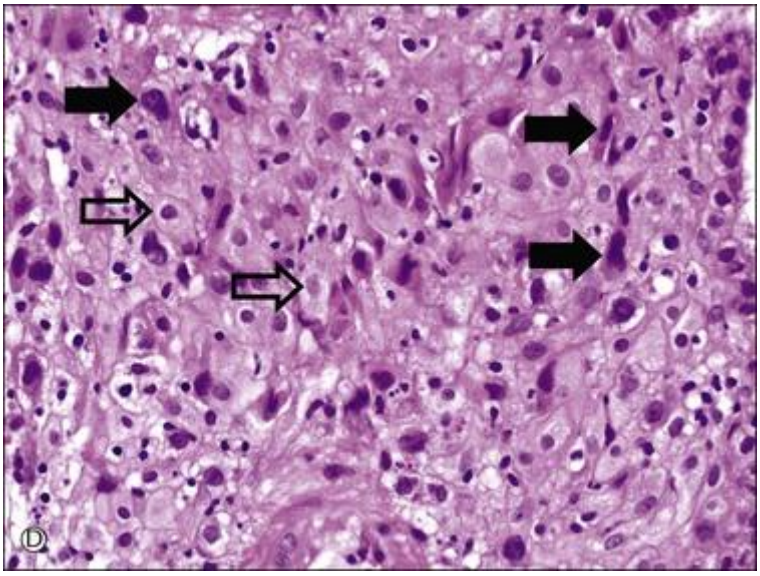


Figure 7 Extra villous trophoblast. Solid arrows represent extravillous trophoblast, hollow arrows represent decidua.

Decidua

The changes that occur in the human endometrium in response to the physiological stimuli of pregnancy and implantation of the blastocyst are

called decidualization. The stromal cells increase in size and number. Blood vessels and glands undergo changes. All these changes are dependent on progesterone [13]eventually assuming an epithelioid appearance. The cells are round to polygonal, with sharply defined cell borders with abundant cytoplasm filled with lipid and glycogen[14], a single nucleus containing a small but prominent nucleolus. During decidualization, the endometrial glands initially enlarge and can often be observed in first trimester specimens. When pregnancy ensues, the nuclei undergo endomitosis, become polyploid, and acquire the morphologic features known as the Arias–Stella change.[15]Ultimately, the glands atrophy, although occasional remnants may be found in the basal plate and in the placental bed.

Fibrinoid

Fibrinoid is a nonfibrous, acellular, relatively homogeneous material derived from cellular secretion, cellular degeneration. Its light microscopic appearance varies from glossy and homogeneous to lamellar, fibrous, or reticular. Because fibrin, blood clot, and secretory products are usually deposited in proximity and cannot be easily discriminated, the general term fibrinoid, rather than fibrin, is used. Fibrinoid is typically found in the following locations:

• Subchorionic region (Langhans' stria)

• Intervillous space (perivillous),

• Chorionic villi (intravillous) ,

• Placental septa

• Cell islands

• Cell columns

• Basal plate

Superficial basal plate, facing the intervillous space (Rohr's stria)

HYPERTENSIVE DISORDERS OF PREGNANCY

(i) Gestational hypertension,

(ii) Preeclampsia

(iii) Eclampsia.

(iv) Chronic hypertension

(v) Chronic hypertension with superimposed pre eclampsia

Gestational hypertension

It is characterised by BP ≥140/90 mm Hg for the first time in pregnancy after 20 weeks, without proteinuria.

Pre eclampsia

Preeclampsia is a multisystem disorder of unknown etiology characterized by development of hypertension to the extent of 140/90 mm Hg or more with proteinuria

after the 20th week in a previously normotensive and nonproteinuric woman.[16] The root cause of preeclampsia is the placenta

Lack of physiologic conversion

During normal placental development, trophoblast invade the maternal spiral arteries, decidua and superficial myometrium, destroy the walls of the arterioles, and replace them with fibrinoid. These converts the vessels into a low resistance, high flow system and renders the vessels incapable of a vasoconstrictive response to the various vasoactive mediators. In women with pre-eclampsia, adequate trophoblast invasion does not occur. This results in inadequate placental perfusion. The aetiology may be of immunological in origin.

The second phase of pre-eclampsia is characterized by widespread endothelial damage leading to platelet adhesion and thrombosis

The principal pathologic changes of the placenta in preeclampsia are:

Grossly the placenta is small placenta, less than the 10th percentile for gestational norms. There may be numerous placental infarcts. Placental abruption may be present.

Microscopically, the placental parenchyma can exhibit a array of sublethal ischemic patterns: villous hypermaturation, increased syncytial knots, distal villous hypoplasia, agglutinated terminal villi, increased cytotrophoblast/fibrinoid islands, and increased perivillous fibrin. Lethal parenchymal ischemia is manifested by placental infarction, with or without abruption. Decidual arteriopathy, when present, ranges from mild (non-transformation of spiral arterioles) to severe (fibrinoid necrosis with or without acute atherosis) (figure 8 & 9).

Chronic maternal vascular malperfusion sufficient to result in severe placental growth restriction (less than the fifth percentile for gestational age) almost invariably manifests significant fetal growth restriction as well. If a sufficient volume of the placental parenchyma is compromised by infarction, abruption, or villous ischemia, hypoxic fetal death results.

DECIDUAL VASCULOPATHY:
• Lack of physiologic conversion • Thrombosis • Atherosis • Fibrinoid necrosis of the media

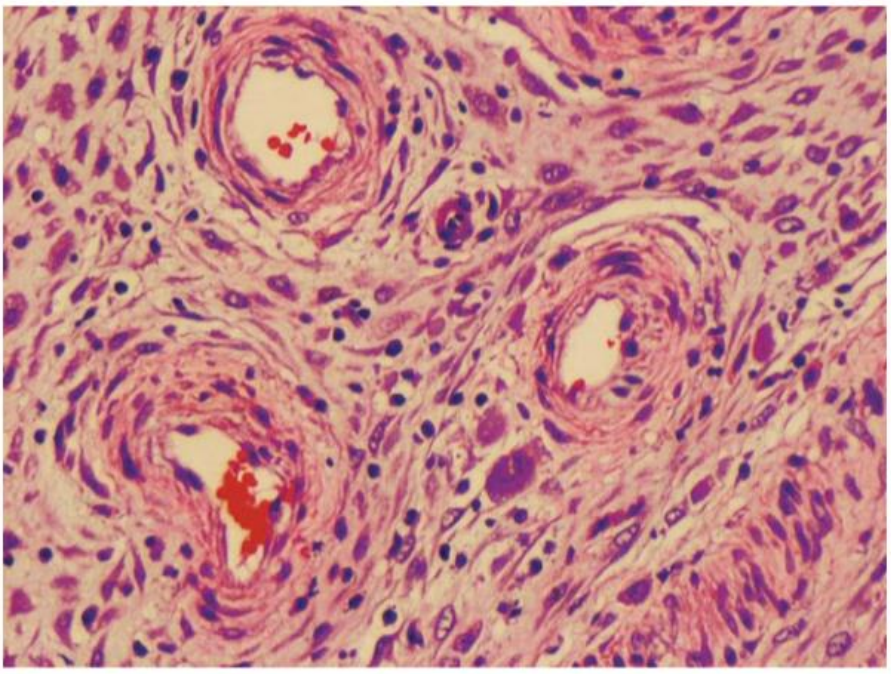


Figure : 8 The arterioles retain their thick muscular coat

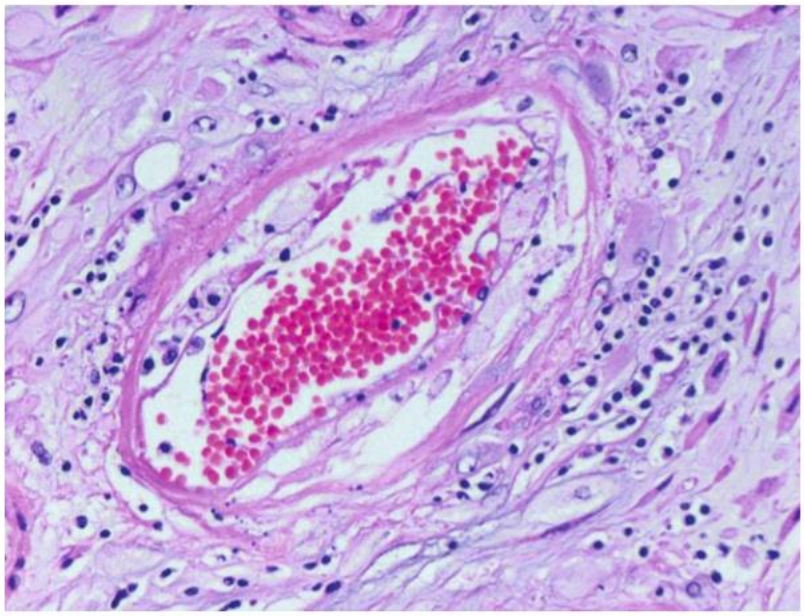


Figure 9: Acute atherosis and fibrinoid degeneration.

Eclampsia is a generalized seizure that occurs during pregnancy in association with features of preeclampsia.

ANAEMIA

Maternal anemia causes hypoxia due to reduced oxygen availability in the intervillous space. Placental villi that develop in hypoxic condition show increased share of fetal capillaries, with no specific signs of proliferation and changes are the result of capillary dilatation.[17]

In hypoxic condition, placental blood vessels continue to develop with the branching mode of angiogenesis to the end of pregnancy, that results in short terminal villi with numerous cross sections of blood capillaries [18]

Severe maternal anemia is characterised by chorangiosis. Chorangiosis is the identification of ten or more vascular channels, in each of ten or more terminal villi, in ten or more ×10 (low-power) fields, in three different areas of non-ischemic placental tissue. Chorangiosis is a manifestation of chronic hypoxia stimulated angiogenesis. Distal villous hypoplasia is seen in severe anemia. Grossly the weight of the placenta is increased in chronic anaemia.

GESTATIONAL DIABETES

Gestational Diabetes Mellitus (GDM) is defined as Impaired Glucose Tolerance (IGT) with onset or first recognition during pregnancy. Worldwide, one in 10 pregnancies is associated with diabetes, 90% of which are GDM. In India, one of the most populous country globally, rates of GDM are estimated to be 10-14.3% which is much higher than the west. Testing for GDM is recommended twice during antenatal period. The first testing should be done during first antenatal contact as early as possible in pregnancy. The second testing should be done during 24-28 weeks of pregnancy if the first test is negative. It is important to ensure second test as many pregnant women develop blood sugar intolerance during this period (24-28 weeks). The threshold blood sugar level of ≥140 mg/dL is taken as cut off for diagnosis of GDM.[19]

Changes include increased placental weight, delayed villous maturation, villous edema, and chorangiosis.[20]

Jarmizek et al reported that most placentas from GDM pregnancies present typical histological findings such as villous immaturity, villous fibrinoid necrosis, chorangiosis, and increased angiogenesis.[21]

PLACENTA ACCRETA, INCRETA, AND PERCRETA

Placenta accreta, increta, and percreta are defined as abnormal adherence of the placenta to the uterine wall so that placental separation does not occur after delivery of the newborn. The degree of abnormal adherence/invasion is variable; placental villi may adhere to (placenta accreta) or invade the myometrium (placenta increta), sometimes penetrating through the serosa (placenta percreta.) The condition may be total (involving the entire placenta), partial (involving one or more cotyledons), or focal (involving isolated foci)[22] Placenta accreta is one of the most common indications for postpartum hysterectomy. Maternal morbidity had been reported to occur in up to 60 % and mortality in up to 7 % of women with morbidly adherent placenta[23]

Risk Factors for placenta accreta are placenta previa[24], repeated cesarean delivery,[25] high parity, and anteriorly low placental location are associated with severe outcome in case of placenta accreta,[26] maternal age

≥35 [27], Prior dilation and curettage of the uterus, Prior myomectomy or other uterine surgery (besides CS), Prior history of accreta, Asherman's syndrome, Prior endometrial ablation.

Jauniaux in 2018 reported that the main cause of placenta accreta spectrum is uterine surgery and, in particular, uterine scar secondary to cesarean delivery. In the absence of endometrial reepithelialization of the scar area the trophoblast and villous tissue can invade deeply within the myometrium, including its circulation, and reach the surrounding pelvic organs.[28]

Histologically, Nitabuch's fibrin and basal villi come into direct contact with the underlying myometrium

Figure : 10 Placenta increta. The placenta has invaded the myometrium almost to the serosa.

Placenta percreta

Characterised by invasion of chorionic villi through the myometrium to the serosa surface of the uterus.

Intrauterine growth restriction

IUGR is defined as fetus that fails to achieve his growth potential. The terms SGA and FGR are not synonymous. It is important to remember that most SGA fetuses are constitutionally small and are not compromised. Intrauterine growth restriction (IUGR) indicates that there is a pathological process operating to restrict the growth rate of the fetus. [29]

Outside of prematurity, IUGR is the second leading cause of perinatal mortality. Mothers who have a history of IUGR with a prior pregnancy are also at risk for recurrence in subsequent pregnancies, including an increased risk of subsequent stillbirth.

Causes of growth restriction may be grouped into maternal, utero-placental, and fetal. Most common cause is utero-placental insufficiency.

Maternal causes

Hypertension, cardiac disease, chronic renal failure, Substance abuse, alcohol, Smoking, autoimmune diseases, poor nutrition

Uteroplacental insufficiency

Pre-eclampsia, placenta accreta, infarction, abruption, placenta praevia.

Fetal causes include genetic abnormalities, congenital abnormalities, congenital infection and multiple pregnancy.[30]

In IUGR, the placenta is usually small. The fetal surface should be examined for vascular thrombosis and the maternal surface for infarcts. The cut surface may show infarcts or increased perivillous fibrin. the umbilical cord is often thin (less than 1 cm in greatest diameter).[31]

Approximately one quarter of placentas associated with FGR lack any morphological abnormality on routine macroscopic and histological examination [32] Changes of uteroplacental insufficiency were the most common finding, being observed in 25% of cases. Of these, one-third had manifestations of early onset pre-eclampsia. Distal villous hypoplasia is characteristically seen in cases of marked intrauterine growth restriction (IUGR) that clinically may be associated with the absence of end-diastolic blood flow in the umbilical arteries. It is characterized by a sparse, poorly developed distal villous tree with abnormally shaped, elongated, slender villi and widening of the intervillous space.[33] Massive perivillous fibrin deposition (≥25% of villi encased by fibrin), is strongly associated with IUGR[34].

Increased knottings for period of gestation are associated with increased risk of fetal intrauterine growth restriction.[35] Villitis of Unknown Etiology is an important cause of intrauterine growth restriction and recurrent reproductive loss. Villitis of Unknown Etiology is caused by maternal T lymphocytes, predominantly CD8-positive, that inappropriately gain access to the villous stroma.[36] Sato et al. noted higher prevalence of placental infarction, fetal vessel thrombosis, and chronic villitis in IUGR compared with normal growth pregnancies

Abruptio placenta

Separation of the placenta—either partially or totally—from its implantation site before delivery is called abruptio placenta.[37] Most common cause of abruption is pregnancy induced hypertension / chronic hypertension. Other causes are trauma, previous abruption, preterm rupture of membranes, thrombophilia[38.] The fresh, acute hematoma is soft, bright red, and can easily be separated from the maternal surface. Within approximately 24 h of the formation of the hematoma it may compress the maternal surface and basal parenchyma. The presence of a shallow, crater-like depression on the maternal surface bordered by congested chorion villous parenchyma, therefore, is a finding highly suggestive of a site of a previous retroplacental hematoma (RH), even in the absence of an accompanying portion of blood clot. Acute lesions consist almost entirely of

red blood cells, but with aging these degenerate and are replaced by fibrin.[39]The overlying, compressed placental parenchyma shows infarction. The chorion of the free membranes and fetal plate may show hemosiderosis.

Histolopathologic dating of the retroplacental haemotoma is difficult, since organization of a RH is different from organization of intravascular thrombus; there is no fibroblastic ingrowth and the center may remain essentially unaltered for a long time. In acute RH, laminated peripheral strands of fibrin can be seen, together with a peripheral neutrophilic reaction in the overlying basal plate. Erythrocytes can be aggregated or pale, or exhibit lysis. Acute formations may exhibit no ischemic chorionic villous histopathology, intravillous hemorrhage 'Recent' hematomas may show hemosiderin-laden macrophages in the maternal space and solidification of the laminations. The adjacent chorionic villi show recent to chronic infarction with ghost-like remnants of degenerated villi and intervillous thrombi. Preserved chorionic villi may show fetal normoblastemia. [40]

Oligohydramnios

It is a condition where the amniotic fluid is deficient in amount to the extent of less than 200 mL at term. Sonographically, it is defined when the maximum vertical pocket of liquor is less than <2 cm or when amniotic fluid

index (AFI) is less than 5 cm (less than 5 percentile). AFI between 5 and 8 is termed as borderline Oligohydramnios.

ETIOLOGY

A. Fetal conditions: (i) Fetal chromosomal or structural anomalies (ii) Renal agenesis (iii) Obstructed uropathy (iv) Spontaneous rupture of the membrane (v) Intrauterine infection (vi) Drugs: PG inhibitors, ACE inhibitors (vii) Postmaturity (viii) IUGR

B. Maternal conditions: (i) Hypertensive disorders (ii) Uteroplacental insufficiency (iii) Dehydration (iv) Idiopathic.[41]

Amnion nodosum is associated with conditions that lead to significant, prolonged oligohydramnios. Lack of amniotic fluid leads to degeneration and death of the epithelium. Vernix becomes deposited on the denuded basement membrane in a nodular fashion. Amnion nodosum develops only late in fetal life.[42] Amnion nodosum consists of small, yellowish-white nodules (1–5 mm) over the amnion. They are readily scraped from the fetal surface.The lesions are composed of deposits of vernix caseosa (an admixture of amorphous, fibrillar, eosinophilic sebum, fetal skin squames, and/or lanugo hairs), affixed to the fetal surface. [43]

On microscopic examination of placenta, basement membrane thickening, haemorrhagic vasculitis, thrombotic vasculopathy, syncytial

knots are seen in cases of oligohydramnios. Fetal thrombotic vasculopathy is a placental lesion characterised by regionally distributed avascular villi and is often accompanied by upstream thrombosis in fetal vessels. It is associated with six times increased risk of oligohydramnios[44]

Intrauterine death

The cause of death may be due to abruption, infarction, chorioamnionitis, villous dysmaturity, insufficiency, perivillous fibrin deposits, placental chorangioma, retroplacental haematoma, feto-maternal haemorrhage, placenta praevia, cord accident, hydrops, hypoplasia, cord abnormalities, intervillositis twin–twin transfusion, post/hyper maturity, chromosomal abnormality, avascular villous clusters, fetal growth restriction, endangiopathia obliterans, severe pre-eclampsia, twin pregnancy, immature birth, uterine anomaly, villitis of unknown etiology (VUE), severe villous fibrosis, vascular hypertrophy, small for gestation placenta, neoplastic disorders, increased syncytial knots[45]

The cause of fetal death could be established in 80% of IUD cases. The remaining 20% did not have a demonstrable diagnosis. The most frequent cause of death in this series was in 40% severe toxemia of pregnancy causing a chronic nutritive placental insufficiency[46]. Maternal vascular malperfusion accounted for a large majority of IUD.

Placenta previa

When the placenta is implanted partially or completely over the lower uterine segment (over and adjacent to the internal os) it is called placenta previa.[47]

Major (grade III or IV) - The placenta lies over the cervical os.

Minor (grade I or II) - The placenta lies in the lower segment, close to or encroaching on the cervical os. [48]

In the vaginally delivered marginal placenta previa, the membranes will have no free margin, and the edge of the placenta will frequently be disrupted and hemorrhagic. There are often old clots at this site, varying from firm, laminated, and brown, to friable loose clots or partly necrotic material that is sometimes green or brown. Portions of placenta are occasionally either atrophied or infarcted. Placenta previa is often associated with placenta accreta and then is called placenta previa accreta[49]

GESTATIONAL TROPHOBLASTIC DISEASE

WHO Classification of gestational trophoblastic disease[50]

Neoplasms
Choriocarcinoma
Placental site trophoblastic tumour
Epithelioid trophoblastic tumour
Non-neoplastic lesions
Exaggerated placental site
Placental site nodule and plaque
Molar pregnancies
Hydatidiform mole
Complete
Partial
Invasive
Abnormal (nonmolar) villous lesions

Hydatidiform mole.

Hydatidiform moles are important to recognize because they are associated with an increased risk of persistent trophoblastic disease (invasive mole) or choriocarcinoma. There is wide range of geographical and ethnic variation of the prevalence of the condition. The molar pregnancy is common in Oriental countries—Philippines, China, Indonesia, Japan, India, Central and Latin America and Africa. The highest incidence is in Philippines being 1 in 80 pregnancies and lowest in European countries 1 in 752 and USA being about 1 in 2,000. The incidence, in India, is about 1 in 400.[51] Approximately 15%–35% of all moles are of the partial type.

They represent an abnormal placenta characterized by enlargement of chorionic villi caused by central edema of the stroma. Variable hyperplasia of the villous trophoblastic cells is present, and this hyperplasia may be marked. A complete mole is distinguished from a partial mole by the amount of villous involvement. The edema is generalized in a complete mole, whereas in a partial mole, the edematous change affects some of the villi. The hydropic villous changes may be related to retarded vasculogenic differentiation in villous stroma. Thus, impairment of vasculogenesis may result in a lack of vascular drainage and cause progressive accumulation of vesicular fluid and lead to the formation of hydropic villi. The risk of developing persistent trophoblastic disease is higher in complete mole than

in partial mole[52] The most important criterion in predicting prognosis is differentiation between partial and complete moles.

Complete Mole

Complete mole results from fertilization of an egg that has lost its female chromosomes, and as a result the genetic material is completely paternally derived Ninety percent have a 46,XX karyotype stemming from the duplication of the genetic material of one sperm (a phenomenon called androgenesis). The remaining 10% result from the fertilization of an empty egg by two sperm; these may have 46,XX or 46,XY karyotype. In complete moles the embryo dies very early in development and therefore is usually not identified. Patients have 2.5% risk of subsequent choriocarcinoma and 17 – 20 % risk of persistent or invasive mole.

Complete moles have two key features: trophoblastic proliferation and villous edema. Many villi display central cistern formation characterized by a prominent central space that is entirely acellular. Smaller villi usually are present but these, too, are edematous. The villous stroma has a distinctive appearance with a pale blue-grey appearance having widely separated spindle cells beneath the villous surface, and edematous central cistern. Villous stroma of a complete mole contains numerous but inconspicuous CD34-positive blood vessels. This trophoblastic proliferation in complete

hydatidiform mole is circumferential around the villus The trophoblast of a complete mole always displays cytologic atypia. [53]

Partial Mole

Partial moles result from fertilization of an egg with two sperm In these moles the karyotype is triploid (e.g., 69,XXY) or occasionally tetraploid (92,XXXY). The volume of placental tissue is relatively normal, and the grossly vesicular villi are mixed with normal-appearing ones. Fetal tissues are typically present. Partial moles have an increased risk of persistent molar disease, but are not associated with choriocarcinoma.[54]

Microscopic features

(1) two populations of villi (one hydropic and one small and fibrotic),

(2) enlarged villi with central cavitation,

(3) irregular villi with geographic, scalloped borders with trophoblast inclusions

(4)minimal trophoblast hyperplasia (usually focal and involving syncytiotrophoblast)[55]

Central cisterns are less conspicuous than in complete moles. Scalloped outline of the enlarged villi, yields a pattern of trophoblastic

invaginations into the villous stroma appearing as inclusions within the stroma. Invaginations are not exclusive for partial moles; may also be seen in complete mole and nonmolar hydropic abortus.

The presence of nucleated red cells representing functioning villous circulation differentiates partial mole from complete mole.

Table 2 : Differences between partial and complete mole

Feature	Complete	Partial
Karyotype	46,XX, 46,XY	69,XXY, 69,XXX
Embryo/fetus	Absent	Present
Hydropic swelling	Marked; cisterns present All villi involved	Less pronounced and focal Cisterns less prominent Small fibrotic villi can be present
Villous outline	Round	Scalloped
Trophoblastic proliferation	Circumferential; Variable, may be marked	Focal and minimal
Trophoblastic atypia	Often present	Absent
Behavior	17–20% develop pGTD	<4% develop pGTD

Histopathologic evaluation can be enhanced by immunohistochemical staining for p57 expression and by molecular genotyping. p57KIP2 is a nuclear protein whose gene is paternally imprinted and maternally expressed. This means that the gene product is

produced only in tissues containing a maternal allele. Because complete moles contain only paternal genes, the p57KIP2 protein is absent in complete moles[56]

The differential diagnosis of a hydatidiform mole includes early nonmolar pregnancy including trisomy placentas. Morphologic distinction of a hydatidiform mole from an abortus with abnormal villous morphology may be problematic, and there is a considerable degree of interobserver variation in making this distinction.[57]

Takayasu Arteritis[58]

Also called pulseless disease, this is a chronic inflammatory arteritis affecting large vessels. It is associated with abnormal angiography of the upper aorta and its main branches and with upper extremity vascular impairment. Comorbid severe renovascular hypertension, cardiac involvement, or pulmonary hypertension worsen pregnancy prognosis. Hypertension is relatively common and should be carefully controlled. Blood pressure is most accurately measured in the lower extremity. Overall, the prognosis for pregnancy is good. Complication includes preeclampsia, preterm birth, and fetal growth restriction or death (Comarmond, 2015). Involvement of the abdominal aorta portends worse perinatal outcome (Sharma, 2000).

Multiple Gestations [59]

There are two types of twin placentation, namely monochorionic and dichorionic. Dichorionic placentas are diamnionic (DiDi), whereas monochorionic placentas can be either diamnionic (DiMo) or monoamnionic (MoMo).

Overall, morbidity is higher in twin gestations. The rates are higher in monochorionic versus dichorionic twin pregnancies.

In dichorionic fused placentas, the dividing membrane will contain chorionic epithelium centrally, whether appearing as one continuous ribbon or interrupted by remnants of immature chorionic villi; whereas monochorionic dividing membranes will be comprised solely of amnion epithelium surfacing two layers of amnion stroma on either side.

Separate placental discs indicate dichorionic placentas; these placentas can be assessed as two singleton placentas. Fused discs can be monochorionic or dichorionic. This issue can be resolved by carefully examining the dividing membranes, which are transparent, like cellophane, in monochorionic placentas due to their composition of two amnions only, or they may be thicker and semitranslucent in dichorionic placentas due to two fused chorionic layers between the amnions. Monochorionic twin placenta should be examined for surface vascular anastomoses.

Fetus papyraceous may be seen in multichorionic gestations which is a small flat disc.

Systemic lupus erythematosus

SLE occurs primarily in young women (M:F ratio, 10:1), and it often complicates pregnancy. The disease is accompanied by a variety of circulating antibodies, of which the best known is the antinuclear antibody (ANA). High perinatal mortality is due to placental damage resulting from either immune complex deposition or from ischaemia due to necrotising vasculitis of the placental bed. [60] Lymphocytotoxic antibodies possessing antitrophoblastic activity have been found in about 80% of patients with SLE. Although the placentas of patients with SLE may be normal, more often they show changes that are frequently impossible to differentiate from the lesions of preeclampsia. When both are present, it is thus difficult to know whether the placental pathology is due to preeclampsia or to SLE.

Histologic study of placentas with SLE reveals decidual vasculopathy and/or atherosis (17%), villitis of unknown etiology (28%), and infarcts (18%), which are principally associated with the simultaneous presence of antiphospholipid antibodies. Thrombosis of decidual vessels and ischemic/hypoxic changes are also prominent. One may see intensive

chronic deciduitis with infiltration of the decidual vascular walls by abundant chronic inflammatory cells in which, unlike preeclampsia, plasma cells predominate. Wolf et al showed a case of systemic lupus erythematosus with placenta showing massive infarction. In the spiral arteries of the basal plate of the placenta, lesions of intimal thickening, fibrinoid necrosis, acute atherosis, and intraluminal thrombosis were observed.[61] The decidual vascular lesions result in placental infarcts and abruptios, which in turn result in retarded placental and fetal growth. The villous tissue displays Tenney-Parker changes, the increased syncytial knotting that is best known in preeclampsia and is due to reduced maternal perfusion. Although Tenney-Parker change, infarcts, and other lesions are usually not seen until the third trimester in preeclampsia, in SLE they are seen in the second trimester as well.

MATERIAL AND METHODS

- Study design : Cross - sectional study
- Study area : Tirunelveli Medical college Hospital
- Study population : women with high risk pregnancy

Duration of study: 1 year from commencement of study after clearance from the Institutional Ethical Committee.

- Sample : Placenta extruded during labour vaginalis and placenta removed during caesarean section will be collected in containers with 10 % neutral buffered formalin.

The total number of specimens studied in the present study was 98 placentas which were obtained from high risk pregnancies.

The specimens were collected from the department of obstetrics, Tirunelveli medical college hospital for a period of 1 year and the study was conducted in the Department Of Pathology, Tirunelveli medical college hospital

INCLUSION CRITERIA:

- Placenta of women who are clinically classified as high risk pregnancy

EXCLUSION CRITERIA

- Normal pregnancy

All the cases were within the age group of 18 - 48 years and includes both primigravida and multigravida. Questionnaire Format and Data Collection Format are developed pretested and coded. Confidentiality will be maintained.

Formal consent were obtained from the pregnant women to collect the specimen. Antenatal history were recorded. Placenta extruded during labour vaginalis and placenta removed during caesarean section were examined in a fresh state and are collected in containers with 10 % neutral buffered formalin. Gross examination of the placenta is done as per standard protocol

EXAMINATION OF PLACENTA

1. Gross examination
2. Microscopic examination

Gross examination of placenta[62]

Examine placenta in fresh state.

The distance from the placental margin to the nearest point of rupture is measured. Membranes are examined for completeness, insertion, decidual necrosis, extra-amniotic pregnancy, retromembranous hemorrhage, meconium staining, color, and transparency. 2–3 cm wide section of membranes beginning with the point of rupture and extending to and including a small portion of placental margin is taken and rolled so that amniotic surface remains inward, and after fixing for 24 hours, 3 mm section is taken from the center. Second section is taken including amnion, chorion, and decidua from the rim of the site of rupture. Remaining membranes are trimmed from the placental margin.

The length of the cord and the shortest distance from the cord insertion to the placental margin are measured. The cord is examined for insertion (nonmembranous or membranous; if latter, are vessels intact?), number of umbilical vessels (by sectioning the cord transversely at two or more points), color, true knots, torsion, stricture, hematoma, thrombosis. Cord is removed from the placenta 3 cm proximal to the insertion, and sections are taken from the cord.

The fetal surface is examined for color, opacity, subchorionic fibrin, cysts, amnion nodosum, squamous metaplasia, thrombosis of fetal surface vessels, chorangioma.

The maternal surface is examined for completeness, normal fissures, laceration, retroplacental haemorrhage.

Maximum diameter, thickness in the center, shape weight are measured (after trimming cord and membranes).

Transverse cuts are made through the maternal surface at 1-cm to 2-cm intervals sparing the fetal surface. Four 2 cm pieces that include the fetal surface and intact maternal surface, selecting tissues of the placenta (within 2 cm of placental margins) are removed and fixed for 24 hours. One section should include the chorionic plate in an area with minimal subchorionic fibrin. The other sections should include the maternal surface. The disk is Examined for infarcts, intervillous thrombi; perivillous fibrin deposition, pallor, consistency, calcification, cysts, tumors. Describe lesion location (central, lateral, or marginal), depth (parabasal, intermediate, or subchorionic), and age of the infarct (recent or old). Photographs are taken

Sections for histology

1. Placenta disk
2. Membranes
3. Cord

Tissue bits are then processed through series of dehydration steps and embedded in wax blocks. Tissue sections of 5 microns thickness are cut from each block and stained by haematoxylin and eosin stain. The different histological findings are observed and quantified.

TISSUE PROCESSING:[63,64]

Tissue processing is a technique which is used for the removal of all extractable water from the tissue and replacing it with a support medium which provides sufficient rigidity to the tissue to enable its sectioning without parenchymal damage or distortion. This is generally achieved by immersion in increasing strengths of ethyl alcohol (ethanol), and is known as dehydration. Since alcohol and wax are not miscible, the alcohol must be replaced by a wax solvent, and since the majority of wax solvents have the effect of raising the refractive index of tissue, which makes them appear clear, this stage has become known as clearing. Finally, there is the impregnation of the tissue with wax, and its casting into a solid block which helps to cut thin sections using rotatory microtome. With each

block 5 micro-meter sections were taken with microtome The slides were placed in incubator at 70^0c for 1 hour.

HEMATOXYLIN AND EOSIN STAIN

Hematoxylin & Eosin stains are the most commonly used stain in histopathology. Hematoxylin is a naturally available basic dye and is extracted from the core or heartwood of the tree Haematoxylon Campechianum and stains the nucleus of the cells, while eosin is an acid xanthene or phthalein dye which is a counterstain and gives a pleasant contrast to the nuclear stain.

The stains used were Harris' hematoxylin and eosin Y

Procedure

Sections are placed in xylene for 1—2 minutes to dissolve the wax. The sections are rehydrated by transferring to absolute alcohol for 1 minute,90 per cent alcohol for 1 minute. Slides are transferred to haematoxylin where they are left for 10 minutes.

Slides are washed until the sections are blue which takes about 10 minutes in tap water of pH 8. Sections are dipped into acid alcohol for a few seconds and then returned to the slide-washing tray until blue again. Sections are transferred to one percent eosin Y for 1 minute. Sections are transferred

from the eosin to the slide-washing tray for 3—4 minutes; After draining, sections are dehydrated by transferring to 90 per cent alcohol for 10—15 seconds, absolute alcohol I for 10-15 seconds, absolute alcohol II for 30 seconds.

Sections are transferred to xylene I and left until completely clear. And then to xylene II from which they may be mounted with DPX

All the slides were viewed under 40x, 100x and 400x view using light microscope. For each case, 100 terminal villi were assessed for maturity and the presence of infarct, syncytial knot, villous edema, villous fibrosis, fibrinoid necrosis, intravillous fibrinoid, perivillous fibrinoid, intervillous haemorrhage, smooth muscle proliferation of feeding vessels, atherosclerosis, thrombosis, villous enlargement, villous vascularity, trophoblastic proliferation, trophoblastic atypia, trophoblastic invasion into myometrium were recorded

RESULT

I studied 98 placentas of high risk pregnancies.

Graph no. 1 – Age distribution of high risk pregnancies

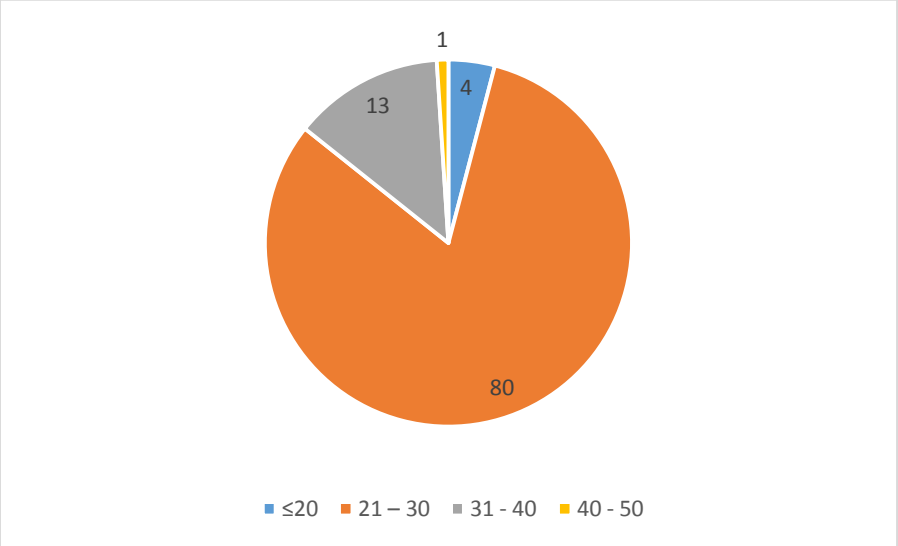


Table 3: Age distribution of high risk pregnancies

Age distribution	NO. OF CASES
≤ 20	4
21 – 30	80
31 - 40	13
40 - 50	1
Total no. of cases	98

The above pie chart shows the age distribution of cases in high risk pregnancies with majority of the cases i.e. 80 cases were between the age of 21-30 years, 13 cases were in the age group of 30 – 40 years, 4 cases were in the age group of ≤20 years of age, one case was in the age group of 40 – 50.

Graph no. 2 – Parity

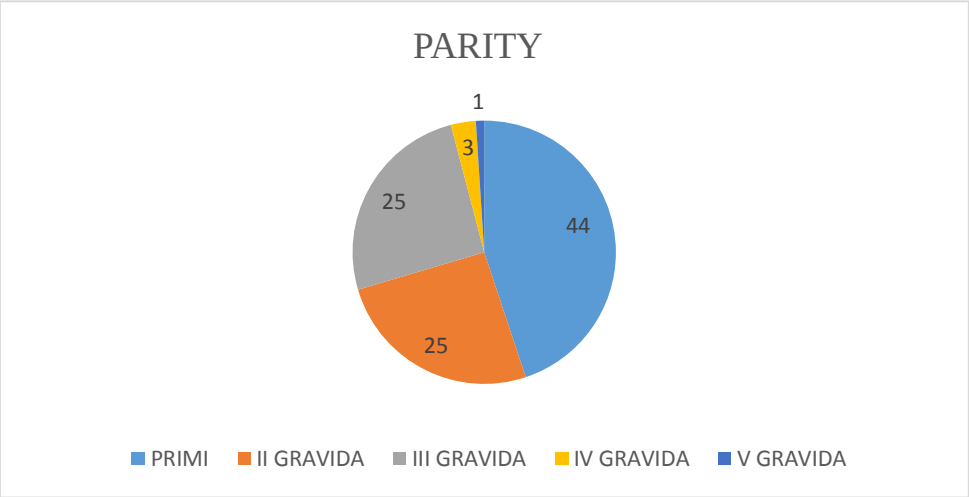


Table 4 : Parity

Parity	Frequency
Primi	44
II Gravida	25
III Gravida	25
IV Gravida	3
V Gravida	1
Total no. of cases	98

Graph 2 shows that 43% of the cases studied were primi, 27.5 % cases were II gravida, 25.5 % cases were III gravida, 3 % cases were IV gravida, 1% cases were V gravida.

Table 5 : Distribution of cases according to risk factors.

RISK FACTORS	FREQUENCY
HYPERTENSIVE DISORDERS OF PREGNANCY	16
ANAEMIA	15
INTRAUTERINE DEATH	13
OLIGOHYDRAMNIOS	11
PLACENTA CRETA SYNDROME	13
PLACENTA PREVIA	8
PARTIAL MOLE	17
COMPLETE MOLE	4
MULTIPLE PREGNANCY	2
IUGR	2
TAKAYASU ARTERITIS	1
SYSTEMIC LUPUS ERYTHEMATOSUS	1
GESTATIONAL DIABETES	5
TOTAL NO. OF CASES	98

Graph no. 3 – Parity and its association with risk factors

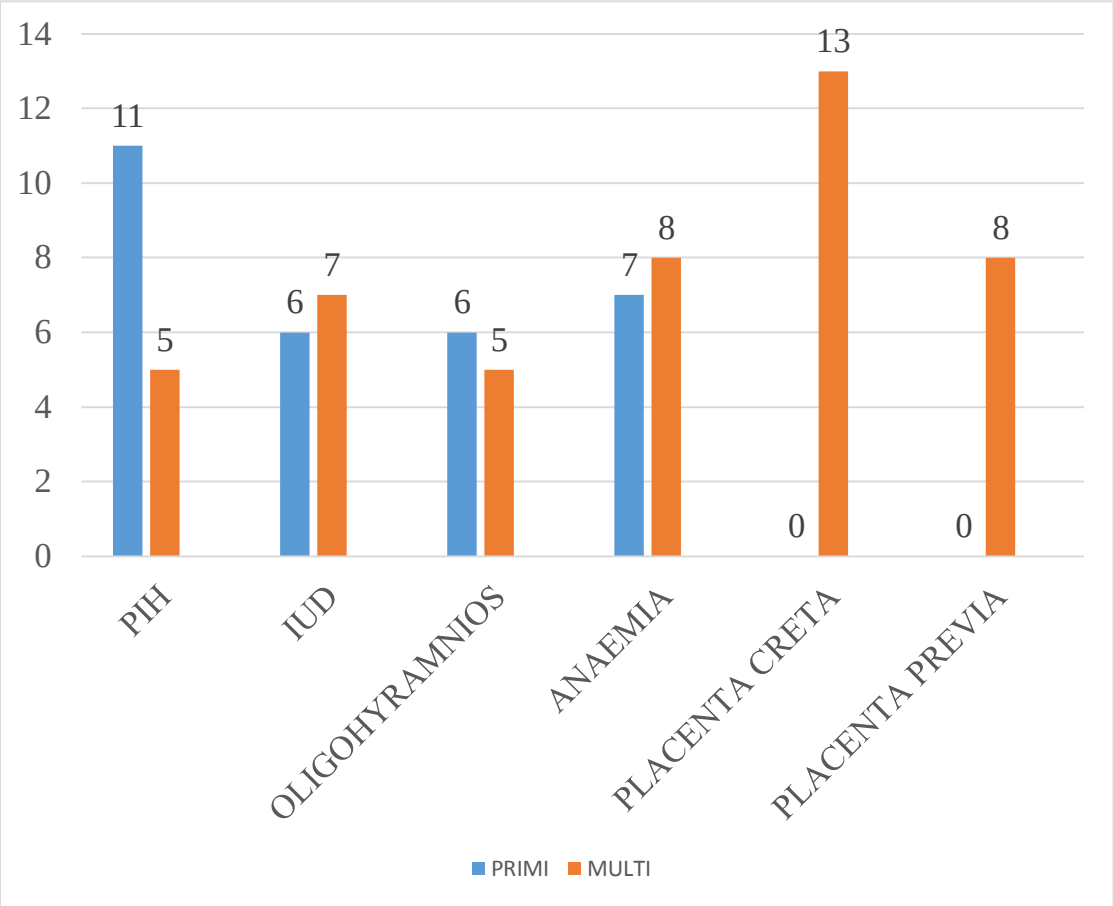


Table 6 : Parity and its association with risk factors

RISK FACTORS	PRIMI	MULTI
PIH	11	5
IUD	6	7
OLIGOHYRAMNIOS	6	5
ANAEMIA	7	8
PLACENTA CRETA	0	13
PLACENTA PREVIA	0	8

From this study it is evident that PIH is more common in primi. Placenta creta and placenta previa occurs only in multigravida.

HYPERTENSIVE DISORDERS OF PREGNANCY

Graph no. 4

Distribution of hypertensive disorders of pregnancy

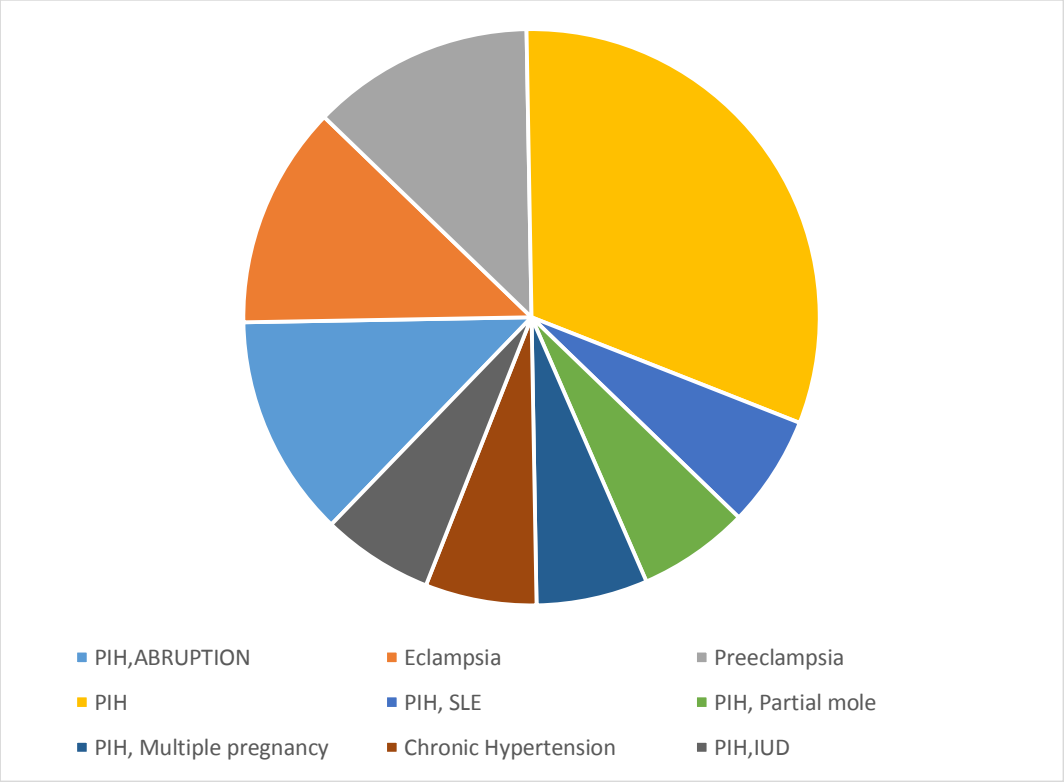


Table : 7 Distribution of hypertensive disorders of pregnancy

RISK FACTORS	NO. OF CASES
ABRUPTION, PIH	2
Eclampsia	2
Preeclampsia	2
PIH	5
PIH, SLE	1
PIH, Partial mole	1
PIH, Multiple pregnancy	1
Chronic Hypertension	1
PIH,IUD	1
Total no. of cases	16

Graph 5

HISTOMORPHOLOGICAL CHANGES IN PLACENTAS OF HYPERTENSIVE DISORDERS OF PREGNANCY

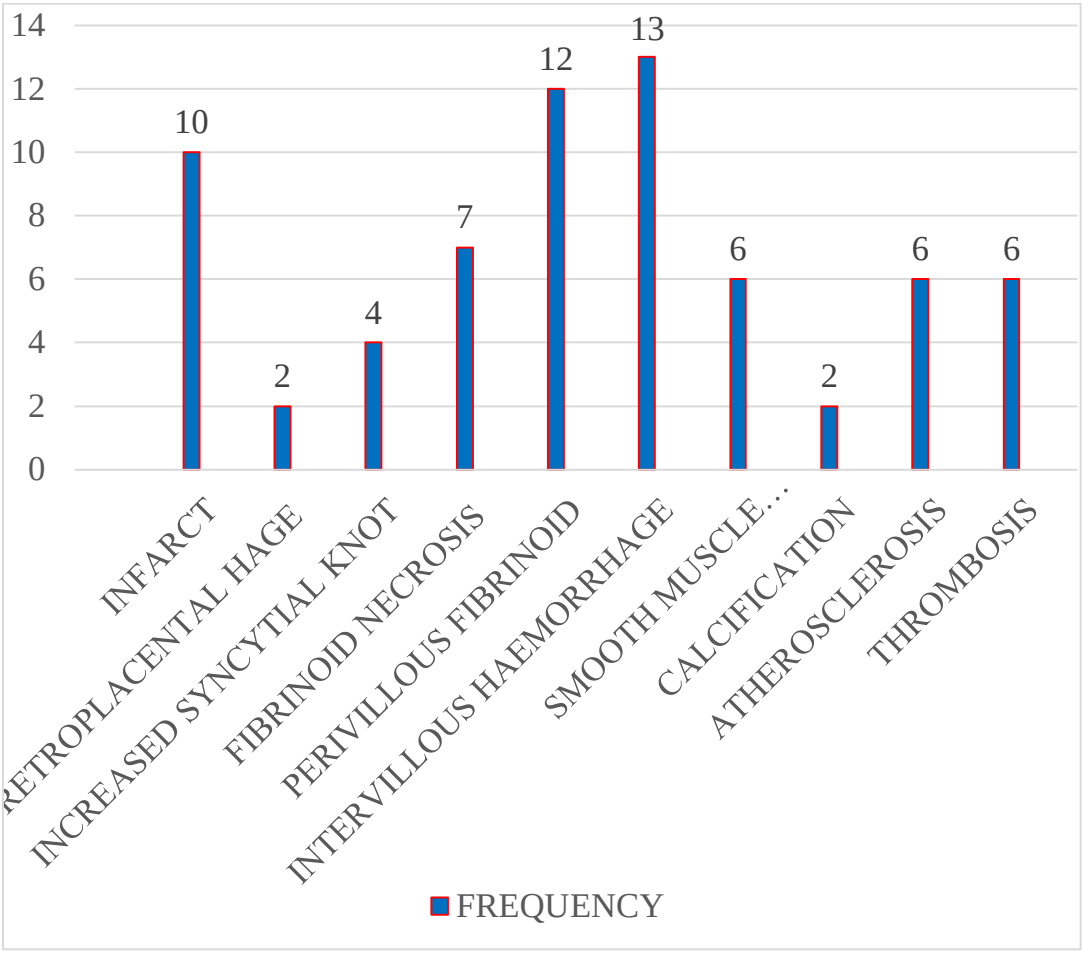


Table : 8 Histomorphological changes in placentas of hypertensive disorders of pregnancy

HISTOMORPHOLOGICAL FINDINGS	**FREQUENCY/16**
INFARCT	**10**
RETROPLACENTAL HAGE	**2**
INCREASED SYNTITIAL KNOTTING	**4**
FIBRINOID NECROSIS	**7**
PERIVILLOUS FIBRINOID	**12**
INTERVILLOUS HAEMORRHAGE	**13**
SMOOTH MUSCLE PROLIFERATION	**6**
CALCIFICATION	**2**
ATHEROSCLEROSIS	**6**
THROMBOSIS	**6**

Out of 16 placentas studied from hypertensive disorders of pregnancy, grossly 10 placentas (62.5%) show infarction. 2 placentas (12.5%) show retro placental haemorrhage (Figure11). 12 placentas (75%) show excessive fibrin deposition, 2 (12.5%) placentas show calcification.

Microscopically 10 placentas (62.5%) show infarction (Figure 12). 2 placentas (12.5%) show retro placental haemorrhage, 4 placentas (25%) show increased syncytial knotting , 7(43.8%) placentas show fibrinoid necrosis, 12 (75%) show perivillous fibrinoid, 13(81%) placentas show intervillous haemorrhage. Six (37.5%) placentas show smooth muscle proliferation of the feeding vessels, six (37.5%) placentas show atherosclerosis (Figure 13), six (37.5%) placentas show villous thrombosis (Figure14). 2 (12.5%) placentas show calcification. One hydatidiform mole is associated with pregnancy induced hypertension. One twin pregnancy case is associated with PIH. One case of IUD is associated with PIH. One case of Takayasu arteritis with chronic hypertension is also included in the study which showed numerous plasma cells and hofbaeur cells in the villous stroma. One case of Systemic lupus erythematosus with chronic hypertension is studied.

Graph 6

HISTOMORPHOLOGICAL CHANGES IN PLACENTAS OF ANAEMIA

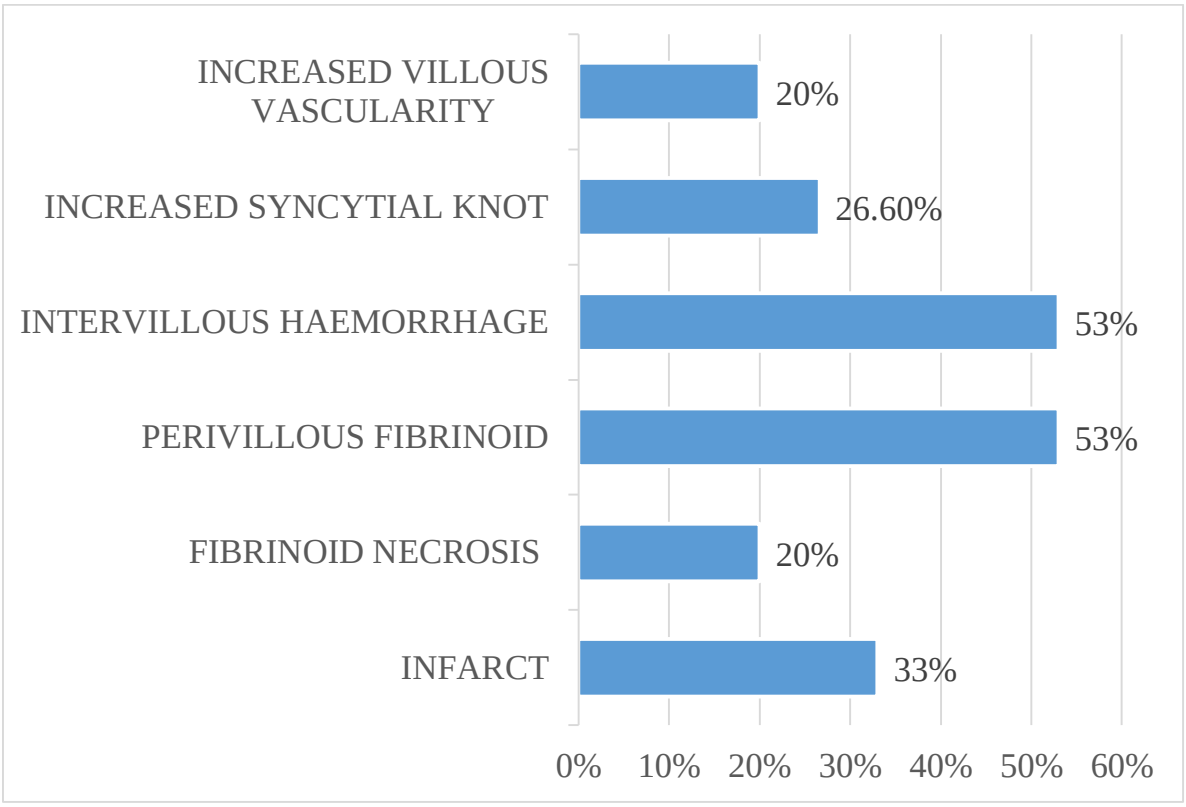


Table 9 : Histomorphological changes in placentas of anaemia

TOTAL NO. OF ANAEMIA	15	100%
INFARCT	**5**	33%
FIBRINOID NECROSIS	**3**	20%
PERIVILLOUS FIBRINOID	**8**	53%
INTERVILLOUS HAEMORRHAGE	**8**	53%
INCREASED SYNCYTIAL KNOT	**4**	26.6%
INCREASED VILLOUS VASCULARITY	**3**	20%

Out of 15 placentas of anaemia studied, five showed infarct, three showed fibrinoid necrosis, eight showed perivillous fibrionoid, eight showed intervillous haemorrhage, four showed increased syncytial knot, three showed increased vascularity of villi, one showed chorangiosis. (figure 15)

Graph 7

HISTOMORPHOLOGICAL CHANGES IN PLACENTAS OF INTRA UTERINE DEATH

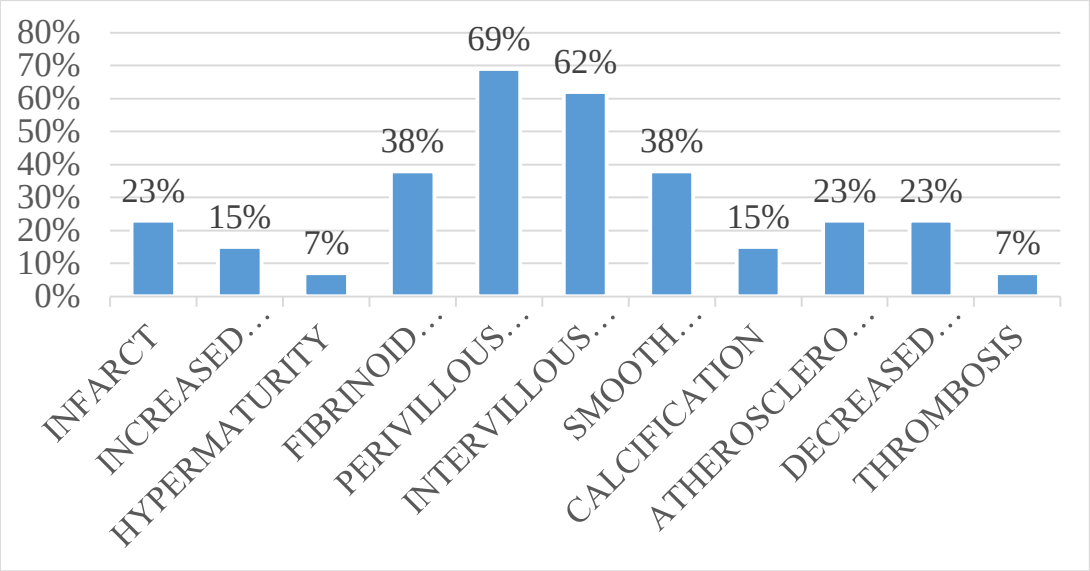


Table 10 : Histomorphological changes in placentas of intra uterine death

TOTAL NO. OF IUD	13	100%
INFARCT	3	23%
INCREASED SYNCYTIAL KNOT	2	15%
HYPERMATURITY	1	7%
FIBRINOID NECROSIS	5	38%
PERIVILLOUS FIBRINOID	9	69%
INTERVILLOUS HAEMORRHAGE	8	62%
SMOOTH MUSCLE PROLIFERATION	5	38%
CALCIFICATION	2	15%
ATHEROSCLEROSIS	3	23%
DECREASED VILLOUS VASCULARITY	3	23%
THROMBOSIS	1	7%

Out of 13 placentas of intrauterine death studied 3 show infarct, 2 show increased syncytial knot, 1 show hypermaturity, 5 show fibrinoid necrosis, 9 show perivillous fibrinoid (Figure 16), 8 show intervillous haemorrhage, 5 show smooth muscle proliferation, 2 show calcification, 3 show atherosclerosis, 3 show decreased villous vascularity, 1 shows thrombosis.

Graph 8

HISTOMORPHOLOGICAL CHANGES IN PLACENTAS OF OLIGOHYDRAMNIOS

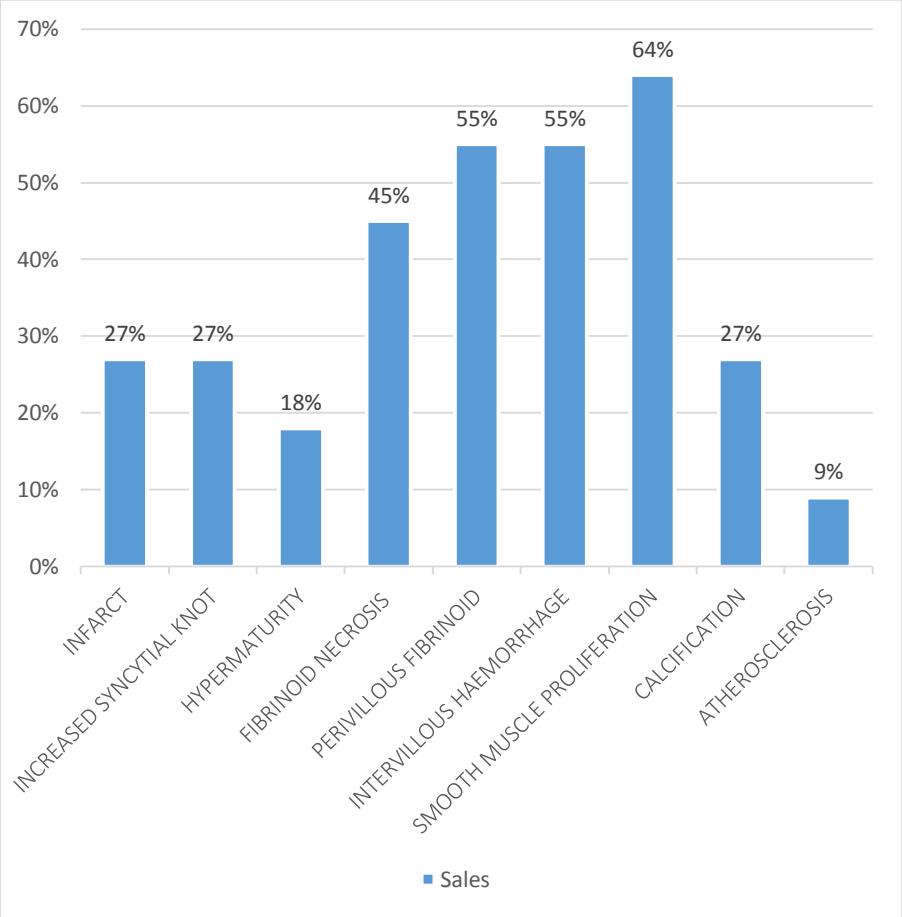


Table : 11 Histomorphological changes in placentas of oligohydramnios

TOTAL NO. OF CASES	11	100%
INFARCT	3	27%
INCREASED SYNCYTIAL KNOT	3	27%
HYPERMATURITY	2	18%
FIBRINOID NECROSIS	5	45%
PERIVILLOUS FIBRINOID	6	55%
INTERVILLOUS HAEMORRHAGE	6	55%
SMOOTH MUSCLE PROLIFERATION	5	45%
CALCIFICATION	3	27%
ATHEROSCLEROSIS	1	9%

Out of 11 placentas studied from oligohydramnios, studied 3 show infarct, 3 show increased syncytial knot (Figure 18), 2 show hypermaturity, 5 show fibrinoid necrosis, 6 show perivillous fibrinoid, 6 show intervillous haemorrhage, 5 show smooth muscle proliferation, (figure17) 3 show calcification, 1 shows atherosclerosis.

Graph 9

ASSOCIATION BETWEEN PLACENTA CRETA AND PLACENTA PREVIA

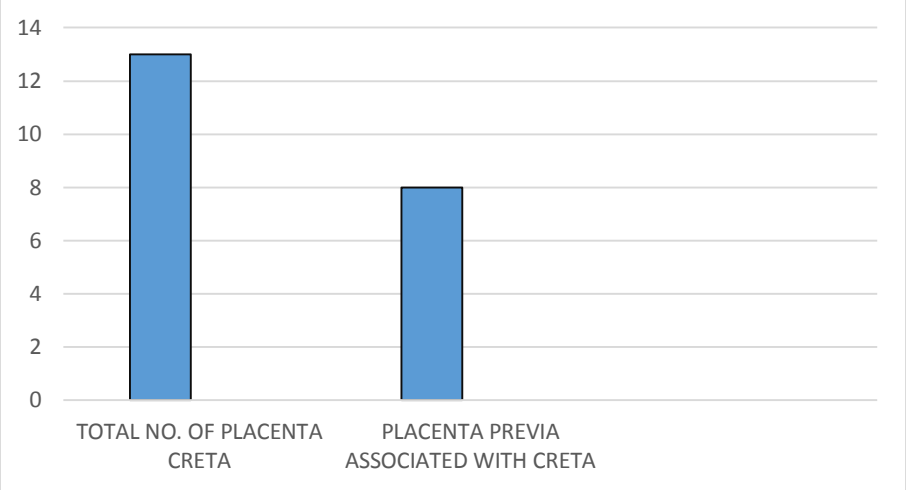


Table 12 : Association between placenta creta and placenta previa

Total no. of placenta creta	13
Placenta creta associated with previa	8

Graph 10

Age distribution of placenta creta

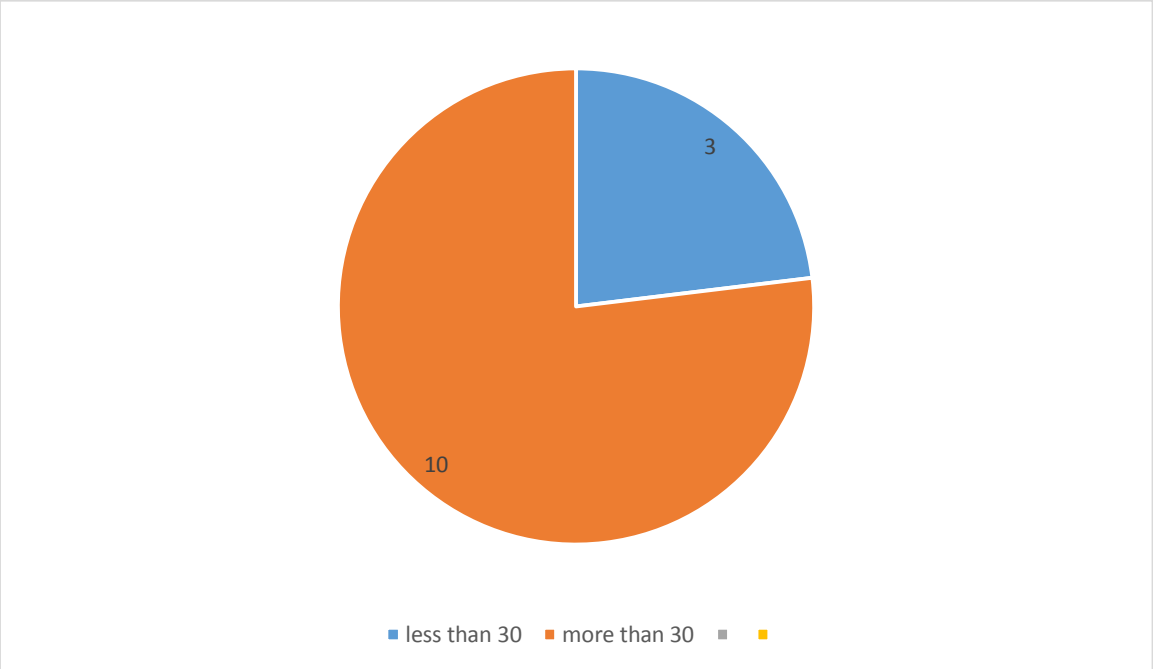


Table 13: Age distribution of placenta creta

Less than 30 years	More than 30 years
3	10

Graph 11

DISTRIBUTION OF PLACENTA ACCRETA, INCRETA & PERCRETA

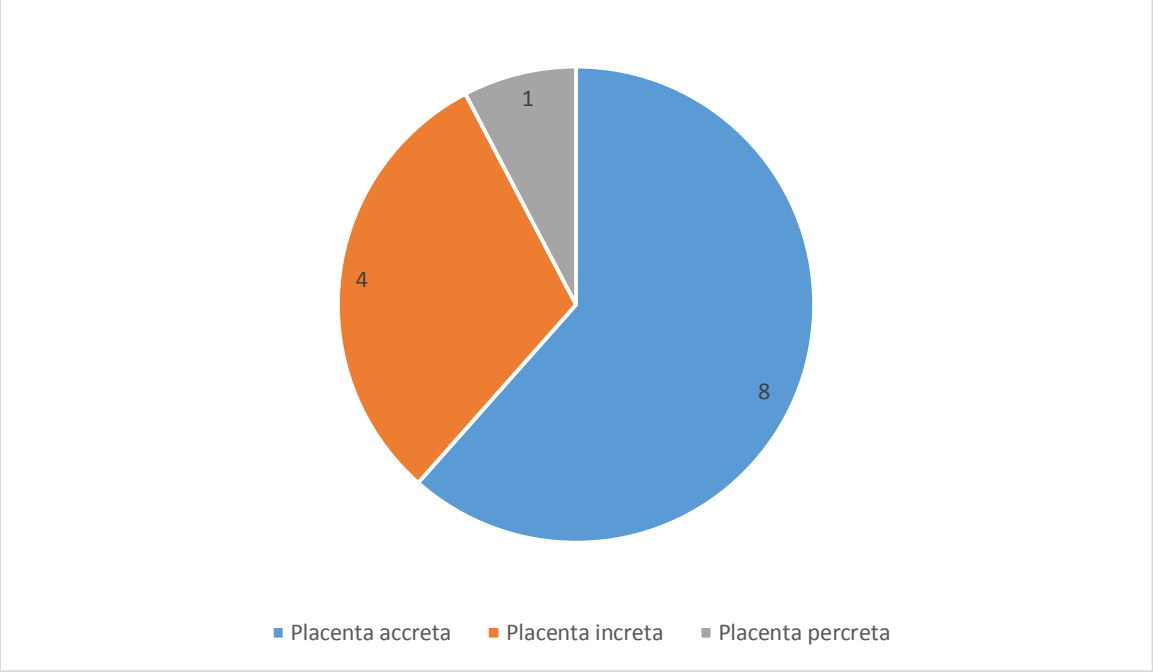


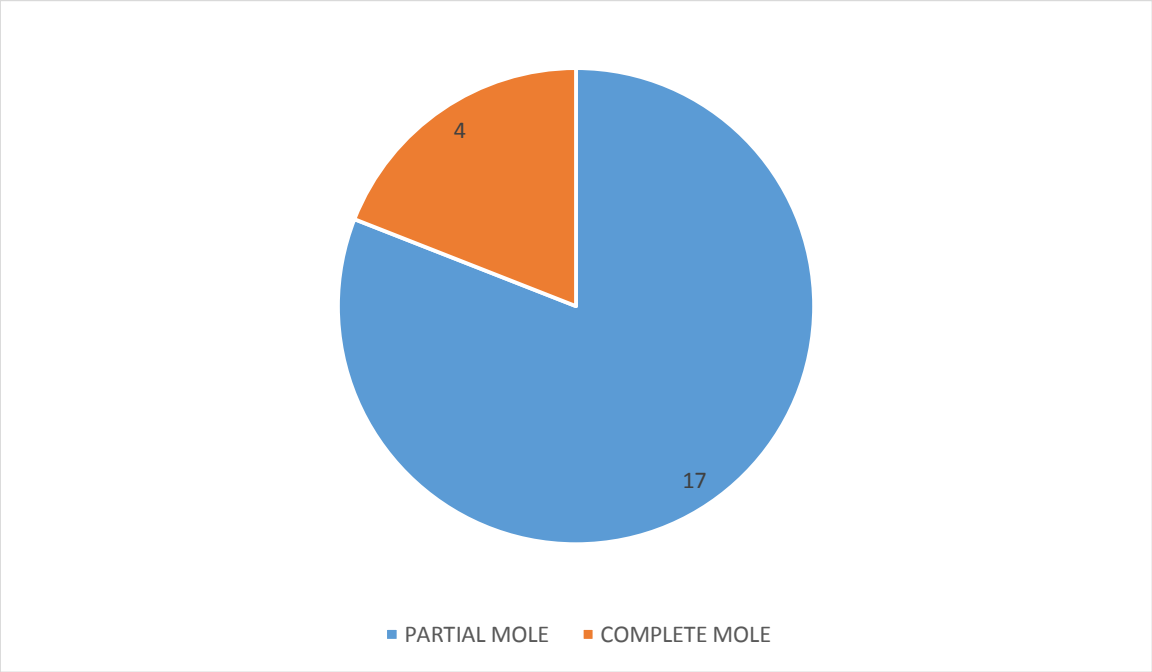
Table 14: Distribution of placenta accrete, increta and percreta

Total no. of creta cases	13
Placenta accreta	8
Placenta increta	4
Placenta percreta	1

Out of 13 cases of placenta creta studied, 8 placentas had the predisposing factor of placenta previa. All 13 placentas are from multigravida patients. Placenta creta is more common in elderly individuals. 8 cases were placenta accreta, 4 cases were placenta increta, one case was placenta percreta. Placenta accreta is more common than increta and percreta. In placenta accreta the placenta is adherent to the myometrium without underlying decidua, whereas in placenta increta (figure 19), the placenta invades the myometrium. In placenta percreta placenta invades the serosa.

Graph 12

DISTRIBUTION OF PARTIAL MOLE AND COMPLETE MOLE



Out of 21 cases of hydatiform mole studied, 17 cases shows partial molar degeneration, 4 cases show complete molar degeneration. 17 cases are in the age group of 20 – 30. 2 patients are in the age group of >30. Two patients are in the age group of < 20.

In partial molar pregnancy there is focal proliferation of trophoblast (figure 21), in complete mole there is circumferential proliferation of trophoblast. In both of these groups there is prominent cistern formation (figure 22). In partial mole the villi are scalloped with the formation of inclusions. In complete mole the villi are predominantly round in shape.

Out of 17 cases of partial mole 6 cases shows absent villous vascularity. Villous vascularity is reduced in 11 cases of partial mole.

Out of 4 cases of complete mole, 3 cases shows absent villous vascularity, 1 case showed reduced villous vascularity.

Trophoblastic atypia

Out of 4 cases of complete mole, 3 (75%) show diffuse moderate trophoblastic atypia, out of 17 cases of partial mole, 5 cases (29%) show focal mild trophoblastic atypia.

PLACENTA PREVIA

Out of 12 cases of placenta previa 8 placentas show features of placenta creta. In our study it occurred only in multigravida. Out of 12 placentas one shows retroplacental haemorrhage. 3 placentas are central plecenta previa. 9 placentas are marginal placenta previa. One out of 12 placentas show infarct and increased perivillous fibrin deposition.

SYSTEMIC LUPUS ERYTHEMATOSUS

One case of placenta from patient suffering from systemic lupus erythematosus is studied. The patient was a primigravida. Placental weight was 400 grams, shows infarction, marginal cord insertion. Also shows Tenney parkar change.

TAKAYASU ARTERITIS

One placenta of takayasu arteritis studied shows poorly developed villi with numerous plasma cells and prominent hoffbaeur cells in the villous stroma. Numerous avascular villi seen. The patient blood pressure during the time of admission was 190/100 mm of Hg.

MULTIPLE PREGNANCY

Out of two cases of multiple pregnancy studied two placentas are monochorionic diamnionic. One patient had pregnancy induced hypertension. The placenta weighed 750 grams and showed increased perivillous fibrin deposition and intervillous haemorrhage. Other patient had gestational diabetes. The placenta weighed 775 grams showed infarct, increased villous vascularity, showed increased fibrinoid necrosis and perivillous fibrin deposition, showed focal villous edema.

INTRAUTERINE GROWTH RESTRICTION

Two placentas of patients diagnosed as IUGR were studied. One from primi, other from III gravida. Both the placenta weighed 400 grams. One showed infarct, increased fibrinoid necrosis, increased intervillous haemorrhage, increased syncytial knotting, increased villous vascularity. Other showed distal villous hypoplasia (Figure 23), dysmature villi, smooth muscle proliferation of the feeding vessels, and hypovascular villi.

GESTATIONAL DIABETES

Out of 5 cases of gestational diabetes studied one is associated with twin pregnancy. The mean weight is 591 grams. Two placentas show infarct, one shows increased vascularity, one shows dysmature villi, three placentas show fibrinoid necrosis, three show increased perivillous fibrin deposition. One shows smooth muscle hyperplasia of the feeding vessels. Two showed increased villous vascularity, one shows stromal villous fibrosis.

Figure : 11 Abruptio placenta showing retroplacental clot.

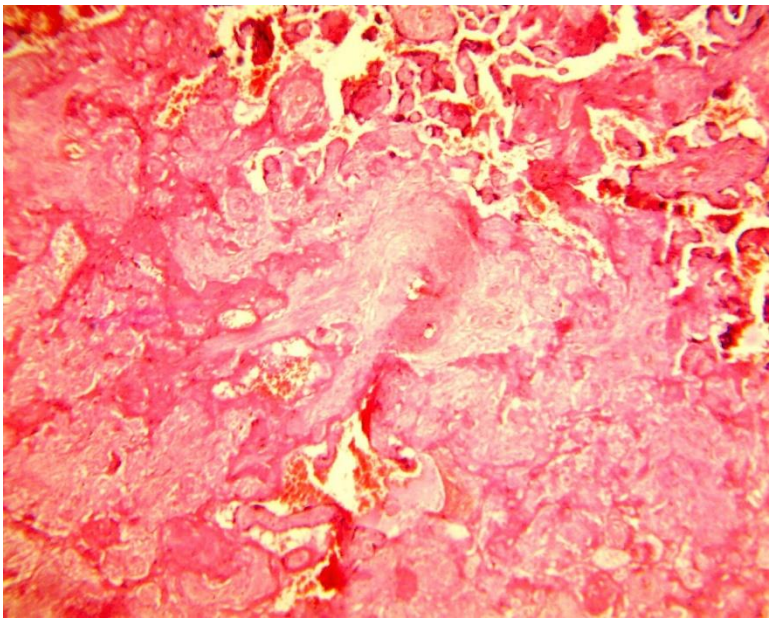


Figure 12: Infarct (Haematoxylin & eosin stain; 10 x view)

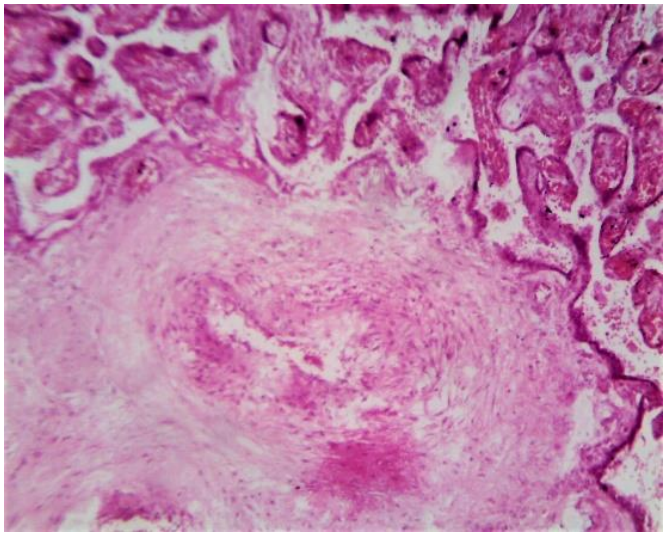


Figure 13: Atheromatous changes and fibrinoid degeneration (Haematoxylin & eosin stain; 10 x view)

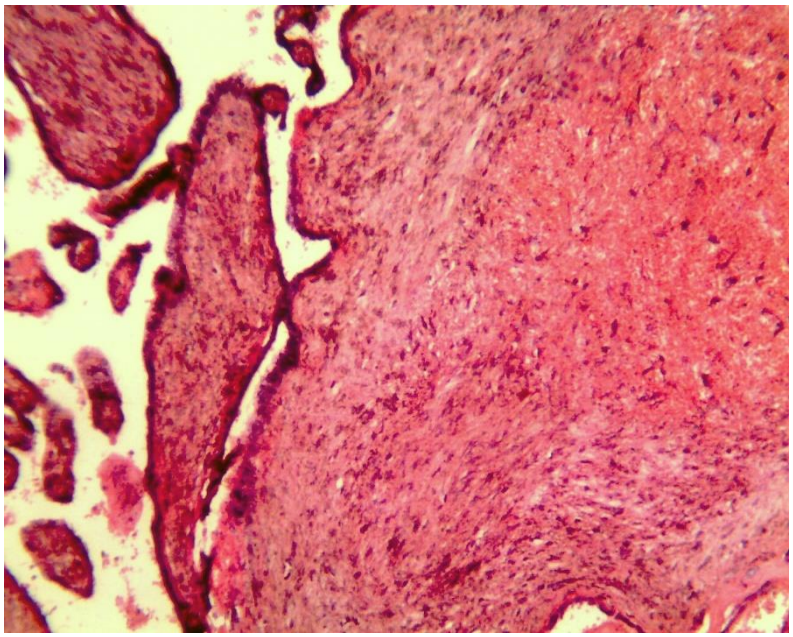


Figure 14 : villous thrombosis (Haematoxylin & eosin stain; 40 x view)

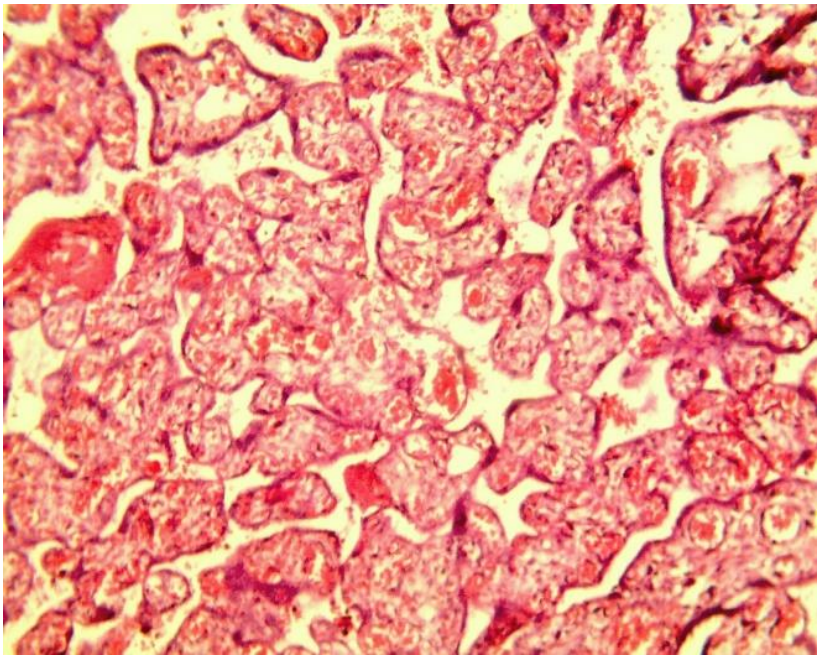


Figure 15: Chorangiosis (Haematoxylin & eosin stain; 10 x view)

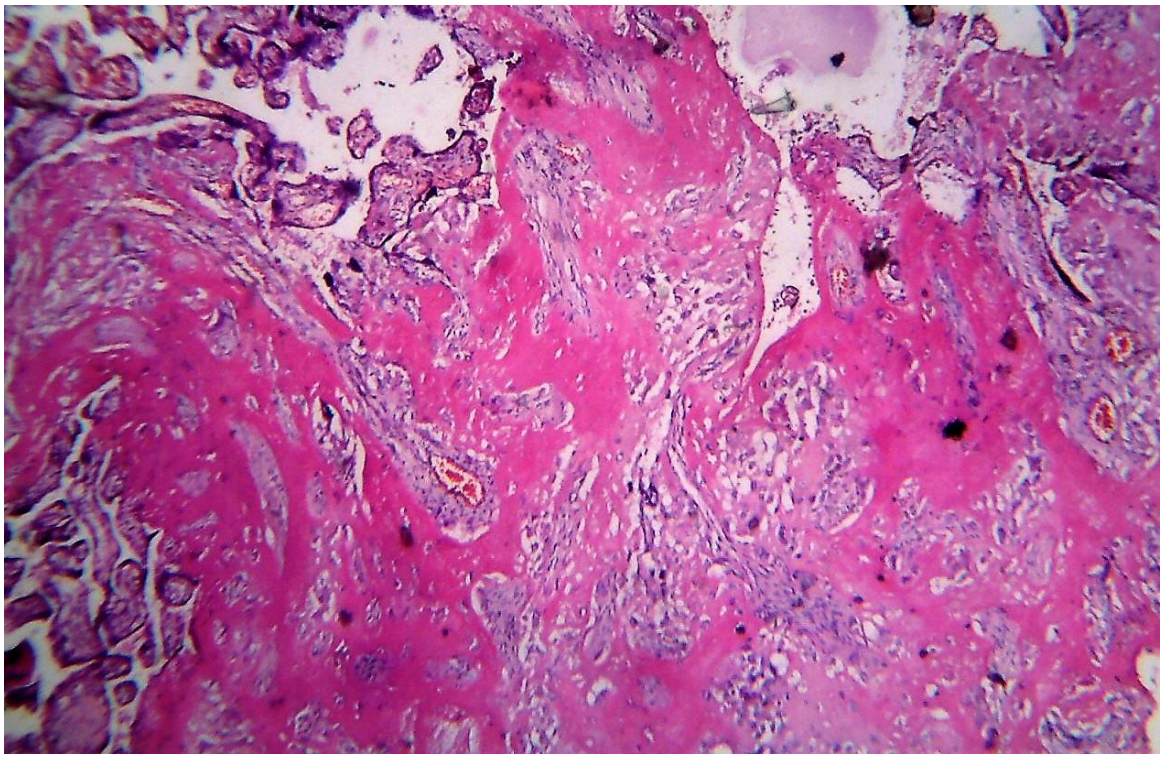


Figure 16: Extensive perivillous fibrin deposition

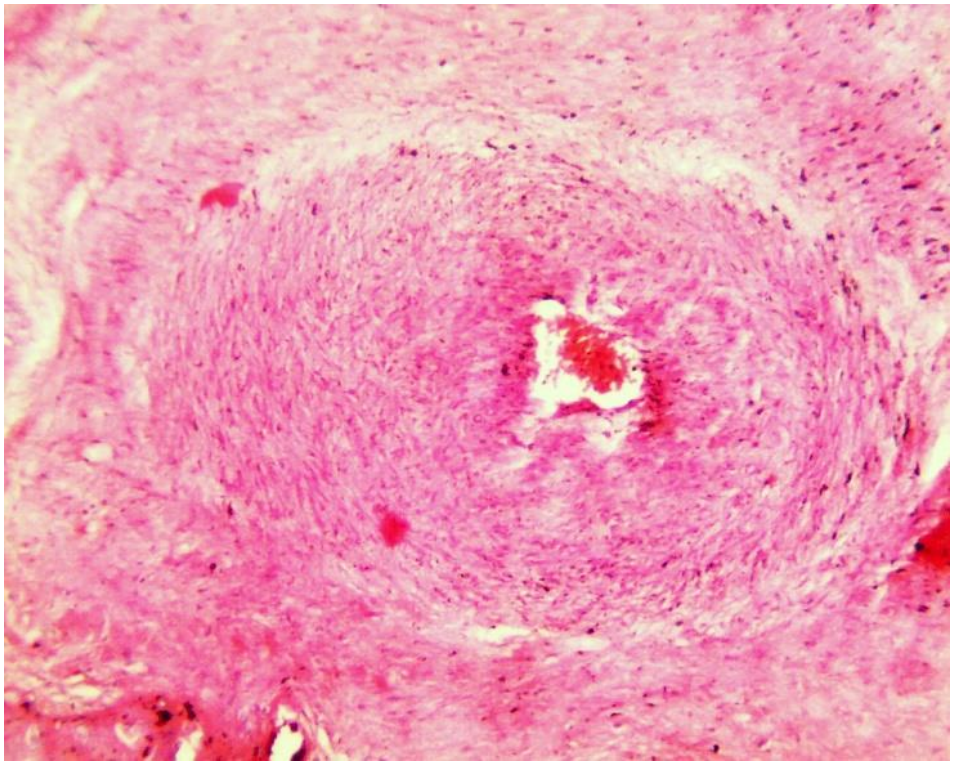


Figure 17: Smooth muscle proliferation of feeding vessels (Haematoxylin & eosin stain; 10 x view)

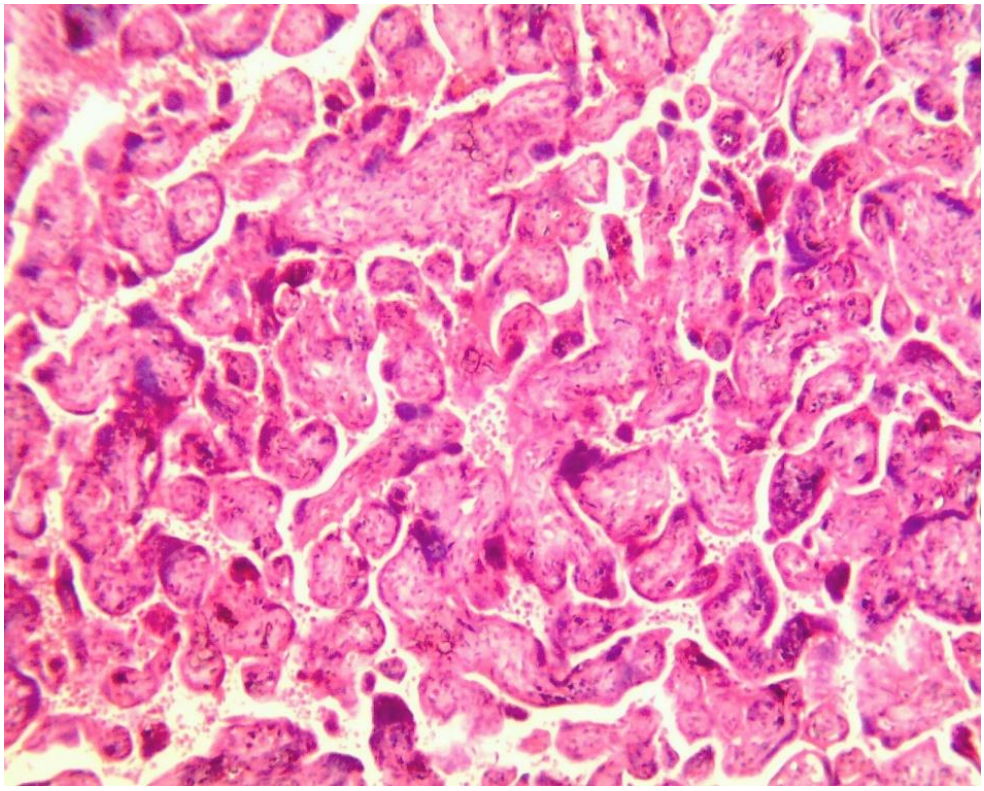


Figure 18: Increased syncytial knotting

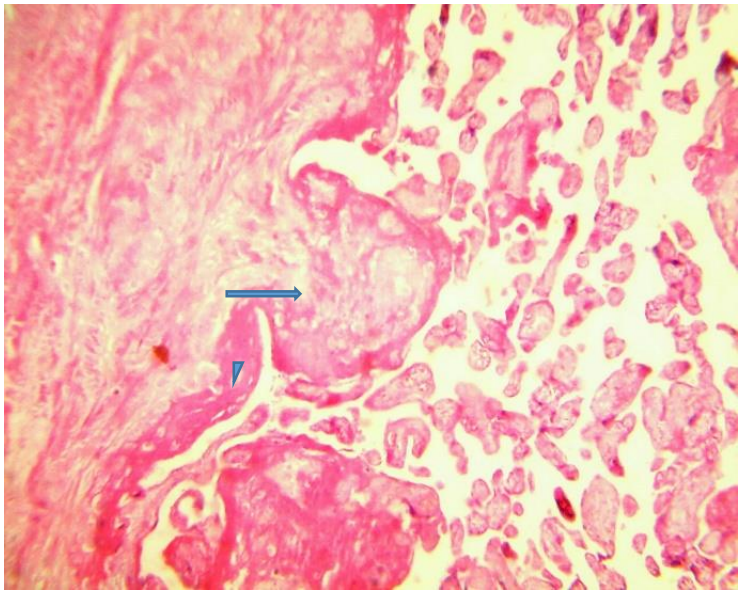


Figure: 19 Placenta increta. The arrow represents the myometrium. The arrowhead represents the Nitabuchs layer of fibrinoid. There is no underlying decidua.

Figure 20 : Placenta showing grape like vesicles

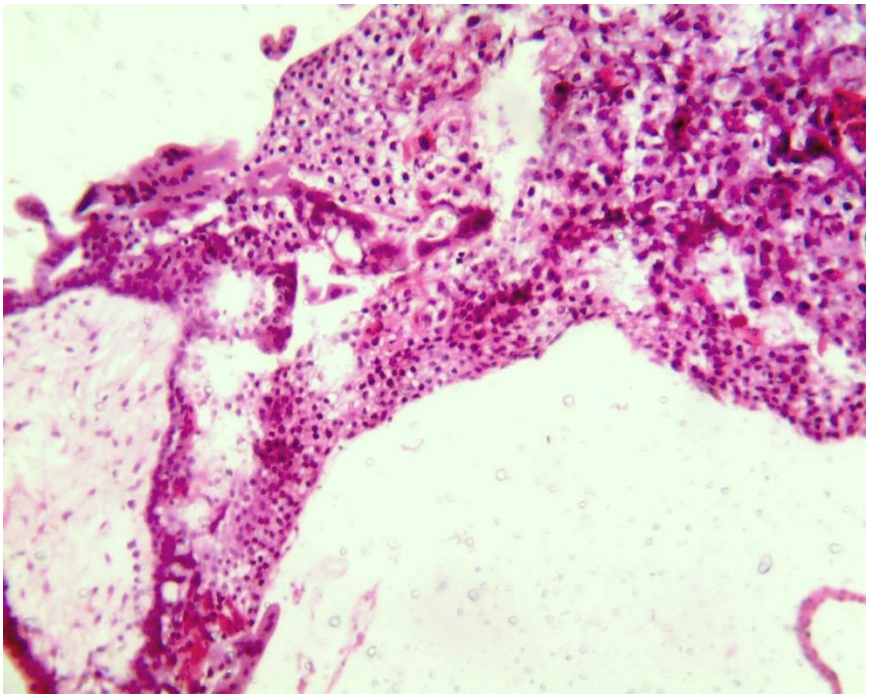


Figure 21 Partial molar pregnancy showing focal trophoblastic proliferation

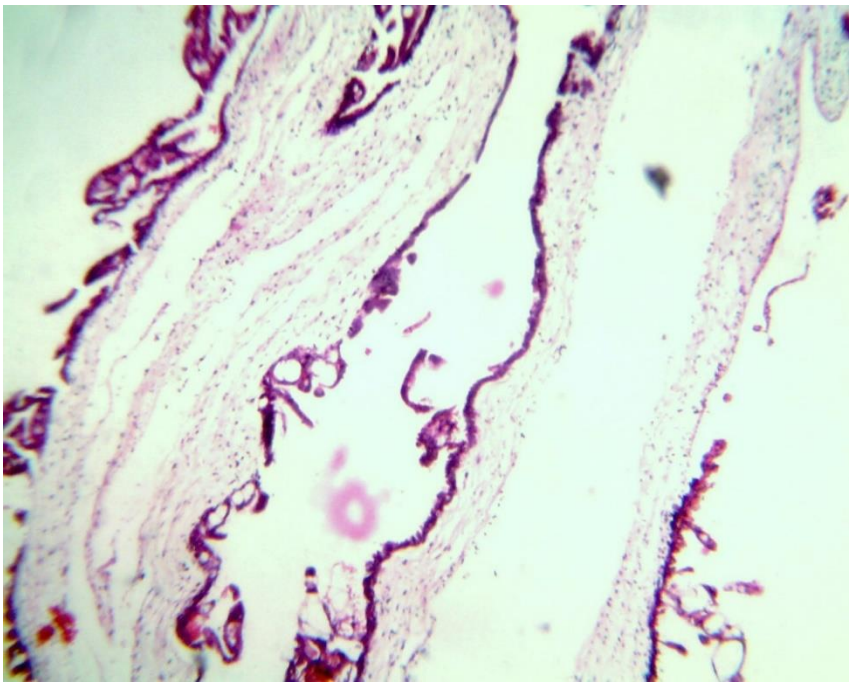


Figure 22: Complete molar pregnancy showing prominent cistern formation Villous vascularity

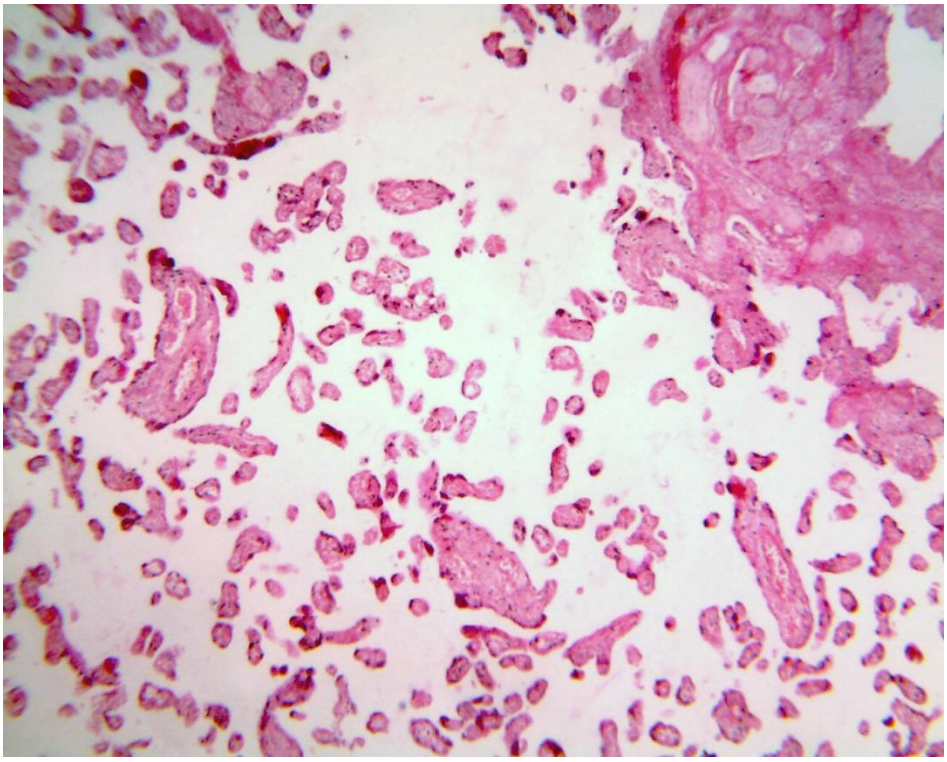


Figure 23: Distal villous hypoplasia (Haematoxylin & eosin stain; 4 x view). Sparse, poorly developed distal villous tree with abnormally shaped, elongated, slender villi and widening of the intervillous space

DISCUSSION

This cross sectional study was carried out in the Department of Pathology, Tirunelveli Medical College, in collaboration with the Department of Obstetrics & Gynaecology, Tirunelveli Medical College Hospital.

A total of 98 specimens were taken for this study which included 16 placentas from patients diagnosed as hypertensive disorders of pregnancy, 15 placentas from patients diagnosed as maternal anaemia, 13 placentas from intrauterine death, 11 placentas from oligohydramnios, 5 placentas from diabetic mothers, 2 placentas from intrauterine growth restriction, 2 placentas from multiple pregnancy, 17 placentas from partial mole, 4 placentas from complete mole, 13 placentas from placenta accreta syndrome.

1) Age of the mother:

In our study the cases showed age ranging 18 years to 48 years.

Above the age of 30 years we have 14 cases, of which placenta creta & intrauterine death are common.

This is in agreement with the study of Kathryn & Susan who reported that placenta creta is more common in elderly woman.[65]

Ling Huang & Sauve found that greater maternal age was significantly associated with an increased risk of stillbirth[66]

2) Parity:

In our study also, 68.7% of placentas from hypertensive groups were primigravida. According to Parth & Shaila from his study of 80 cases of placenta, reported that hypertensive disorders of pregnancy is more common in primigravida.[67]. This has also been observed by Debi Prasad & Sumita Tripathy[68]

In our study 100% of placenta previa and placenta creta are multigravida. This is in agreement with the Shahida Shaikh [69] & Gielchinsky[70]

3) Hypertensive disorders of pregnancy

In the present study we studied 16 placentas from hypertensive disorders of pregnancy, of which 9 cases had gestational hypertension, 4 cases had preeclampsia/eclampsia, 2 cases had abruption, 1 case had chronic hypertension.

According to Sreechithra acute atherosis, fibrinoid necrosis and endothelial proliferation were more specific and significant in pregnancy induced hypertension.[71]

Dhabhai et al found increased syncytial knots, hyalinized villi, stromal fibrosis and fibrinoid necrosis in cases of PIH[72]

Pradnya Saragade found a significantly increased prevalence of calcifications, syncytial knots, infarcts and fibrinoid necrosis in cases of PIH[73]

We observed increased incidence of infarction, excessive intra villous and perivillous fibrin, increased syncytial knotting, increased intervillous haemorrhage. We also observed smooth muscle proliferation of the feeding vessels, atherosclerosis, thrombosis. Gestational hypertension with abruption showed large retroplacental haemorrhage.

4) Anaemia

In the present study we assessed 15 placentas associated with maternal anaemia (mothers with Hb level < 11 g/dl).

Out of 15 placentas of anaemia studied, we observed increased incidence of perivillous fibrionoid, intervillous haemorrhage, fibrinoid necrosis, increased syncytial knot, infarct.

We also observed increased vascularity of villi. One placenta showed chorangiosis.

Biswas et al, reported thinned out syncytiotrophoblastic linings with increased syncytial knots, increased intravillous fibrin deposition and

perivillous fibrin deposition, infarction, increased number of hypovascular villi.[74]

According to Baske, most frequently noticed changes were fibrinoid necrosis, hyalinised villous spots and villous fibrosis, dilated and congested villous vessels and increased syncytial knots, infarct[75]

J Stanek defines several histologic features of placental hypoxia. These include villous infarcts, villous maturation, villous vascularity, syncytial knots, increased perivillous fibrinoid. Our study is in agreement with the findings of Stanek.[76]

5) Intrauterine death

Out of 13 placentas of intrauterine death studied, the gestational period ranges from 22 weeks to 37 weeks. The majority of stillbirths were associated with some degree of placental abnormality on histological examination.

We observed perivillous fibrinoid and intervillous haemorrhage in majority of the cases.

According to Amrutha et al, intervillous hemorrhage is characterized by breaks in the villous epithelium secondary to ischemia and showed a higher incidence of IUD[77]

We also observed infarct, increased syncytial knot, hypermaturity, fibrinoid necrosis, smooth muscle proliferation, villous vascularity, calcification, thrombosis & atherosclerosis.

Histologically in such cases regressive changes of the placenta, such as microinfarcts, necrosis and deposition of intervillous fibrin were found[78].

Sofiya & Pushpa reported extensive placental infarction, placental insufficiency changes, chorioamnionitis, abruption, extensive perivillous fibrin deposition and hypoplastic placenta[79].

6) Systemic lupus erythematosus

One case of placenta of systemic lupus erythematosus studied. The significant findings were decreased placental weight and infarction. These findings were in agreement with the study of Magid et al.[80] and Hanle et al.[81]

We also observed excessive syncytial knotting which is called as Tenney parker change.

7) Oligohydramnios

Out of 11 placentas studied from oligohydramnios, we observed infarct, increased syncytial knot, hypermaturity, fibrinoid necrosis, perivillous fibrinoid, intervillous haemorrhage calcification. These findings were consistent with the study of Spinillo A[82] et al, Hamadany MZ[83]

Gupta et al. in his 50 cases of oligohydramnios also reported similar findings like intervillous fibrin deposition, calcification, syncytial knots.[84]

We also observed smooth muscle proliferation, atherosclerosis.

8) IUGR

Two placentas of IUGR were studied. We observed reduction in weight of the placenta. This findings are in accordance with the study of Hemalatha & Phani Kumar

Histopathologically we observed infarct, increased fibrinoid necrosis, increased intervillous haemorrhage, increased syncytial knotting, distal villous hypoplasia, smooth muscle proliferation of the feeding vessels. One placenta showed increased villous vascularity, other placenta showed hypovascular villi.

This study was in accordance with the study of Kotgirwar S et al., who reported increased fibrinoid necrosis, increased perivillous fibrinoid deposition, increased syncytial knots and increased placental infarction in placentae in IUGR cases[85]

Hemalatha & Phani Kumar from their study of 75 IUGR placentas also observed increased incidence of infarction, intervillous fibrin deposition, syncytial knots. Hypovascularity of villi were found in some cases.[86]

Intervillous hemorrhage is characterized by breaks in the villous epithelium secondary to ischemia and showed a higher incidence of IUD, IUGR[87]

9) Gestational diabetes

Out of 5 cases of gestational diabetes studied we observed increased placental weight. This is in accordance with the study of Gauster et al,[88] Sharmila et al [89] and Ahmed et al.[90]

In the present study we observed increased incidence of fibrinoid necrosis and perivillous fibrinoid, stromal villous fibrosis in placentas of GDM. This finding is in accordance with the study of Nidhi et al,[91]

We observed increased villous vascularity, dysmature villi, which are in accordance with the study of Huynh et al [92] and Patrycja et al [93]

10. Placenta creta

We studied 13 cases of placenta creta. 62% is associated with placenta previa and is more common in elderly individuals. This is in accordance with the study of Hung et al[94] and Fitzpatrick[95]

Placenta accreta is more common than increta and percreta. This is in accordance with the study of Bartels et al.[96]

11. Hydatiform mole

We studied 21 cases of hydatiform mole. 17 cases shows partial molar degeneration, 4 cases show complete molar degeneration.

Out of 4 cases of complete mole, 3 (75%) show diffuse moderate trophoblastic atypia, out of 17 cases of partial mole, 5 cases (29%) show focal mild trophoblastic atypia.

According to Montes et al, implantation site trophoblastic atypia was predominantly focal in 40% of 30 partial moles (33% mild atypia; 7% moderate-severe atypia); and, predominantly diffuse in 87% of 47 complete moles (21% mild atypia; 66% moderate-severe atypia). [97]

Our study is in accordance with the above study. Thus we conclude that trophoblast of the implantation site exhibits focal, mild atypia in some partial hydatidiform moles, and diffuse marked atypia in most complete hydatidiform moles. Thus, implantation site trophoblastic atypia may be a useful pathologic guideline regarding the diagnosis and classification of hydatidiform moles.

SUMMARY AND CONCLUSION

This study throws light on the morphological and histological changes of placenta in high risk pregnancies. This will help us to understand the etio-pathogenesis of high risk pregnancy. In placentas of intrauterine growth restriction and intrauterine death, examination of placenta will plays a crucial role. In our study placenta creta and IUD are more common in elderly women. Hypertensive disorders of pregnancy are more common in primigravida. Placenta creta and previa are more common in multigravida. The placental weight is increased in gestational diabetes and reduced in IUGR. In most of the high risk cases studied we found perivillous fibrinoid and intervillous haemorrhage as predominant findings. Infarction is most frequently observed in hypertensive disorders of pregnancy. Smooth muscle proliferation of the feeding vessels is predominantly seen in oligohydramnios. Placenta accrete is more common than increta and percreta. Placenta previa is an obvious risk factor for placenta creta syndrome. Implantation site trophoblastic atypia may be a useful pathologic guideline regarding the diagnosis and classification of hydatidiform moles. Examination of placentae will be helpful in preventing the adverse effects in successive pregnancies.